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39.

1150.

A TREATISE

ON THE

DISEASES OF THE HEART

AND

GREAT VESSELS,

AND ON THE

AFFECTIONS WHICH MAY BE MISTAKEN FOR THEM:

COMPRISING THE

AUTHOR'S VIEW OF THE PHYSIOLOGY OF THE HEART'S ACTION AND SOUNDS,
AS DEMONSTRATED BY HIS EXPERIMENTS ON THE MOTIONS AND SOUNDS IN 1830,
AND ON THE SOUNDS IN 1834-5.

BY J. HOPE, M.D. F.R.S.

OF ST. GEORGE'S HOSPITAL; FORMERLY SENIOR PHYSICIAN TO THE ST. MARYLEBONE INFIRMARY;
EXTRAORDINARY MEMBER, AND FORMERLY PRESIDENT, OF THE ROYAL
MEDICAL SOCIETY OF EDINBURGH, ETC.

> "Most of the phenomena which Nature presents are very complicated; and, when the effects of all known causes are estimated with exactness and subducted, the residual facts are constantly appearing in the form of phenomena altogether new, and leading to the most important conclusions."—HERSCHEL, Prelim. Disc., p. 156.

Third Edition, Corrected and greatly Enlarged.

LONDON:
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MDCCCXXXIX.

1150.

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TO

DR. ALEXANDER HANNAY, GLASGOW.

My dear Hannay,

Little less than twenty years have elapsed since we studied auscultation together as house physicians to the Edinburgh Infirmary. At that time, there were few auscultators and many opponents in the land. We have lived to see these circumstances reversed; and to you, whose zeal and talents have contributed so powerfully to the change, it must be gratifying to behold this once suspected department of medical science recognized as one of the greatest of discoveries, cultivated with avidity by all classes of our profession, and—what is still better—extensively alleviating the sufferings of our fellow-creatures.

To you I am indebted for having first drawn my attention to the subject: to you I wish to inscribe this trifling result of my labours, and inadequate testimony of my esteem.

Hinc (omne) principium, huc refer exitum.

Always, my dear Hannay,
Your sincere friend,

J. HOPE.

13, *Lower Seymour-street*,
May 11, 1839.

PREFACE

TO THE THIRD EDITION.

THE addition of one-third of new matter to the present volume, and the care with which the whole has been revised and corrected, will, I trust, sufficiently prove my respect for the favourable opinion of my professional brethren, as evinced, not in this country only, but also on the European and American continents, by the sale of no less than six or seven editions and translations in as many years.

The additions, including, I hope, some useful original matter, though they pervade every part of the work, will be found principally under the following heads:—

1. The natural sounds of the heart.* 2. The sound of costal

---

* My labours on this subject have been appropriated by a certain gentleman, who, however, has not the most remote pretensions to them. He says, in the Med. Gaz. for September 1835, p. 818, "In the last number of the Med. Gaz. there is an account of *my* experiments on the sounds of the heart, extracted from an appendix to Dr. Hope's work on the Diseases of the Heart. The statement of the experiments is correct, as Dr. Hope was present at the *greater part* (the whole) of them, and *I also sent him the original notes.*" For the facts, see p. 30, and the foot-note.

Several writers and reviewers have been led into the error of ascribing these experiments to the individual alluded to: *e. g. Professor Müller*, in the translation of his Physiology, part iv. p. 176, and Appendix. p. 1: *Dr. Alison*, in his Supplement to his Outlines of Physiology, p. 18, 1836, &c.

percussion, with or without tinnitus (Laennec's Cliquetis). 3. Murmurs from valvular disease, and the whole subject of particular valvular diagnosis, which will now, I confidently hope, be found one of the most simple and easy departments of auscultation. 4. Murmurs of the heart and arteries independent of organic disease. 5. Venous murmurs. 6. Musical murmurs. 7. Abdominal murmurs, both connected with pregnancy, and otherwise. 8. Tremor or thrill of the heart, arteries, and veins. 9. Signs, general and physical, of pericarditis and endo-pericarditis. 10. Connexion of diseases of the heart with apoplexy, palsy, &c. 11. Partial dilatation or real aneurism of the heart. 12. The signs, physical and general, and the pulse of softening. 13. Signs of adipose disease of the heart. 14. Aneurisms of the aorta bursting into the pulmonary artery and the right ventricle. 15. Abdominal aneurisms. 16. Anæmic, nervous, dyspeptic, plethoric, bilious, and other sympathetic affections of the heart, with their diagnosis. 17. Displacements. 18. The pulses of disease of the heart.

To the complaints made by some, that additions and alterations so considerable have been so tardily published,* though I have habitually taught most of them to my class and in the hospital for several years, I can only reply by pleading my utter inability, even if there had been the inclination, to devote more than an average share of attention to any one subject;—an inability which rests upon all those who, to the private distractions of a laborious profession, add the onerous duties of hospital physicians and teachers of the practice of medicine. I can, indeed, truly say, with Senac and others, that I have worked slowly and painfully, inter "tædia et labores," in fragments of time hardly redeemed from excessive professional engagements.

I have studied brevity to the utmost, my object always having been, to offer the pith of the whole subject in the smallest possible compass. For this reason, I have avoided the multiplication of prolix cases,—offering no new ones except for the illustration of new points or doctrines. As, however, I have sometimes referred to numbers so large as might seem incredible without explanation, it may be proper to state, once for all, that, out of upwards of 15,000 hospital in- and out-patients, whom I have treated during the last eight years, about seven per cent., as near as I can judge from a rough calculation,* have laboured under organic disease of the heart,—making a total of 1,050 cases, exclusive of those occurring in private practice.

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* Except the experiments on the sounds, published in the Appendix to the previous edition, in April, 1835.

I have in several instances transferred to their proper sources discoveries which, in the first edition, I imagined to have belonged to myself. Any similar oversights in the present edition I shall be happy to rectify, if the authors will oblige me with the information.

I have, on the same principle, and, I trust, with perfect good feeling, ventured to reclaim for my countrymen and self a number of discoveries, which an eminent French writer, probably from unacquaintance with the English language and medical literature, has imagined to have emanated from himself.

Finally, I have added a few plates, which I trust will be found convenient.

I have to express my obligations to Mr. James Freeman for the excellent Alphabetical Index.†

J. H.

13, *Lower Seymour-st. London,*
May 11, 1839.

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* If I have leisure hereafter to sort these cases, I trust to give the exact per centage.

† In an Appendix prefixed to this Index will be found the important autopsies of the cases of V . . . ., Esq., and Goff.

INDEX OF CASES.

A. B., 341
Adipose degeneration, three cases of, 348
A Genevese, 607
——— referred to, 355
Allen, John, 605
A . . . n, Esq., 603
Anderson, Christian, 571
——— referred to, 27, 51, 71, 76, 79, 80, 293, 367, 376, 388
Aneurism of the abdominal aorta, case of, 462
Aneurism situated immediately behind the heart, 447
A . . . . r, Dr., 604
Bowden, Grace, 597
——— referred to, 386
Brown, James, 666
———, referred to, 74, 320, 365
Bryant, Robert, 543
Carrington, 602
C———n, Esq., 516
Collard, Richard, 545
Collins, Mary, 491
———, referred to, 72, 75, 240, 367, 473, 489
Copas, John, 550
———, referred to, 73, 153, 365, 380
Cyanosis, two cases of, 491
Dennis, Elizabeth, 573
———, referred to, 77, 376
Dolan, John, 567
———, referred to, 368, 369, 376, 382
Evans, James, 469
———, referred to, 379, 385
Fenn, Ann, 578
———, referred to, 370
Franklin, James, 135
Gillan, Patrick, 555
———, referred to, 293
Goff, John, 610; autopsy, 626
Green, John, 549
Harrison, William, 579
———, referred to, 194
Hedgley, William, 564
———, referred to, 565, 385
H . . . y, Esq., 77, 308
Hill (see the old editions)
James, Phœbe, 115
———, referred to, 125 (by mistake for Bowden), 385, 537
Jones, Robert, 590
———, referred to, 84, 88, 125, 167, 387, 467, 473
Keith, David, 547
Lafin, 608
———, referred to, 430
Lambert, William, 557
———, referred to, 294
—l—n, Mrs. 576
——— referred to, 78, 79, 293, 364, 388, 397
L— P—, Miss, 595
——— referred to, 76, 84, 125, 386, 452, 491
M . . . . . Miss, 536
Macearl, Henry, 560
Malformation, two cases of, 491
May, Joseph, 580
———, referred to, 150, 153, 194, 366, 403
Milton, Henry, 587
———, referred to, 73, 87, 199
Mitchell, John, 466
———, referred to, 74, 75, 84, 125, 330, 379, 385, 422, 471, 485, 488
Murmurs, three cases of anomalous, 391
N . . . . , Esq., 79
N . . . e, Miss, 605
Palpitation, eight cases of, (see *Contents*, *Palpitation*,) 512
Payne, Benjamin, 569

Payne, Benjamin, referred to, 194, 380
P . . . . . . .d, Esq., 348
Porter, Richard, 562
————————, referred to, 153, 366
P . . . . . . .r, Mr., 351
Pyke, Sarah, 605
R., Lady, 598
————, referred to, 76, 225, 360, 386, 420
R., Master, 492
————————, referred to, 473, 489
Rogers, Edmond, 599
————————————, referred to, 472
R. S., Esq., 28
————————, referred to, 74
Saunders, Mr. Wm., 342
Sharpe, George, 574
————————————, referred to, 79, 367, 376
Sir ——, Bart., 343
S. . . . . . . .n, Esq. 350
Snowden, John, 552
————————, referred to, 153
Softening, cases of, 341
Storer, Richard, 565
Tindall, Joseph, 593
————————————, referred to, 88
Utero-placental murmur, cases of, 134
V. . . ., Esq., 589
————————, autopsy, 625
————————, referred to, 88
W——, Esq. 586
————, referred to, 74, 385
Wetherly, Sarah, 594
————————————, referred to, 2, 76, 125, 367, 386, 452, 472, 491
Williams, Charles, 583
————————————, referred to, 466
Wood, Thomas, 28

CONTENTS.

Introduction xxi

PART I.

ANATOMY AND PHYSIOLOGY OF THE HEART.

Page
CHAP. I. ANATOMY OF THE HEART 1
Situation of the heart 2
Percussion of the heart 5
Causes which prevent dulness on percussion 8
CHAP. II. ON THE ACTION AND SOUNDS OF THE HEART 9
SECT. I. *Experimental Researches on the Action of the Heart* ... 9
Experiments performed on rabbits and frogs, July 31, 1839 11
Experiments, Aug. 21, 1830 13
Conclusions on the motions of the heart 18
Conclusions on the sounds 19
Conclusions on the rhythm 20
Experiments repeated, Aug. 10, 1831 20
SECT. II. *Experimental, Physiological and Pathological Researches on the Sounds of the Heart* 25
Experiments, Series i. 32
———— Series ii. 34
———— Series iii. 35
Conclusions from the whole of the experiments on the sounds 39
a. Conclusions on the first sound 39
How far it is caused by muscular extension 42
How far it is caused by bruit musculaire 45
How far it is caused by extension of the auricular valves 45
b. Conclusions on the second sound 50

Page
c. Conclusions on murmurs artificially produced 50
Erroneous or defective Theories of the Sounds of the Heart 51
Laennec 51
Dr. C. J. B. Williams 52
Mr. Turner 52
Dr. Corrigan 52
M. Pigeaux 53
M. Marc d'Espine 53
Dr. Billing 53
M. Bouillaud 53
Mr. H. Carlile 53
M. Majendie 54
CHAP. III. Physiological Phenomena of the Heart's Action and Sounds founded on the foregoing Researches ... 55
1. The phenomena of the heart's action in the order of their occurrence 55
2. Causes, mechanism and objects of the motions 57
3. Causes and mechanism of the sounds 63
First principle of the heart's motion 65
CHAP. IV. Pathological Phenomena of the Heart's Action and Sounds 67
Sect. I. *Modifications of the Motions and Sounds by Hypertrophy and Dilatation* 67
Simple hypertrophy 67
Simple dilatation 68
Hypertrophy with dilatation 69
Sect. II. *Murmurs produced by Valvular Disease* 70
Aortic valves; 1. Systolic murmur 72
2. Diastolic murmur 73
Pulmonic valves; 1. Systolic murmur 75
2. Diastolic murmur 76
Mitral valve; 1. Systolic murmur 76
2. Diastolic murmur 78
Tricuspid valve; 1. Systolic murmur 79
2. Diastolic murmur 80
Mechanism and varieties of valvular murmur 80
Continuous murmur in the heart 84
Pitch or key of valvular murmurs 85
Musical murmurs in the heart 86
Summary of conclusions on murmurs in the heart ... 89
Situations in which murmurs of the respective valves are most audible 90
Sect. III. *Murmur from Hypertrophy with Dilatation, and its Mechanism* 94
Sect. IV. *Murmurs in the Heart and Arteries independent of Organic Disease* 95
Laennec's views incorrect 95
Author's views, with proofs 98
Experiments on Dogs 100
Recapitulation of causes 102

Page
Author's views applied to murmurs, *a*, from compression of arteries ... 104
b. From loss of blood ... 106
c. From anæmia, &c. ... 106
d. From arterial varix ... 107
Musical murmurs not arterial, but venous ... 108
Sect. V. *Venous or Continuous Murmur, Hum, and Musical Notes* 109
Venous murmur ... 109
Musical venous murmur ... 118
Constitutional causes of venous murmurs ... 122
Sect. VI. *Purring Tremor or Thrill of the Heart and Arteries* ... 124
1. In the heart ... 124
2. In the arteries ... 125
3. Inorganic tremors in the arteries ... 126
Inorganic tremor in the veins ... 605
CHAP. V. Auscultation applied to Pregnancy ... 127
1. The double beat of the fœtal heart ... 127
2. The murmur usually called utero-placental ... 131
Cases illustrative of murmurs occurring in pregnancy, &c. ... 134
Seat of the murmur of pregnancy ... 140
Practical conclusions ... 142

PART II.

Inflammatory Affections of the Heart and Great Vessels ... 143
CHAP. I. On Pericarditis ... 144
Sect. I. *Anatomical Characters of acute Pericarditis* ... 144
1. Preternatural redness of the pericardium ... 144
2. Coagulable lymph adhering to the surface of the pericardium ... 146
Object of adhesion ... 148
Process of adhesion ... 149
Opake white spots on the surface of the heart ... 151
Analogous transformations of false membrane ... 151
3. Fluid effused within the cavity of the pericardium ... 152
Anatomical characters of chronic pericarditis ... 153
Sect. II. *Signs and Diagnosis of acute Pericarditis* ... 154
1. General signs ... 155
Signs of amelioration ... 163
2. Physical signs. Percussion ... 164
Impulse ... 165
Sounds ... 165
Attrition-murmurs ... 166
Murmurs of coexistent endocarditis ... 171
Diagnosis of valvular, from attrition murmurs ... 174
Signs and diagnosis of chronic pericarditis ... 174
General signs ... 174
Physical signs ... 176

Page
SECT. III. *Causes of Pericarditis* 176
SECT. IV. *Progress and Duration, Terminations and Prognosis of Pericarditis* 180
Progress and duration 180
Terminations 181
Prognosis 182
SECT. V. *Treatment of Pericarditis* 184
Treatment of acute pericarditis 184
Treatment of chronic pericarditis 189
SECT. VI. *Adhesion of the Pericardium* 191
Diagnosis of adhesion 193
CHAP. II. CARDITIS, OR INFLAMMATION OF THE MUSCULAR SUBSTANCE ... 196
1. Universal carditis 196
2. Partial carditis, abscess, ulcer, rupture of walls, valves, aorta, &c. 197
CHAP. III. ENDOCARDITIS, OR INFLAMMATION OF THE INTERNAL MEMBRANE OF THE HEART 200
SECT. I. *Anatomical Characters of acute Endocarditis* 203
A. Redness of the internal membrane of the heart and arteries 203
1. Non-inflammatory redness 203
2. Inflammatory redness 208
B. Effusion of lymph on the internal membrane, with thickening of its substance 208
Concretions of blood from endocarditis 211
SECT. II. *Signs and Diagnosis of Endocarditis* 212
General signs 213
Physical signs 215
SECT. III. *Causes, Progress and Duration, Terminations, Prognosis, and Treatment of Endocarditis* 217
Causes 217
Progress, duration, and terminations 217
Prognosis 218
Treatment 219
CHAP. IV. ACUTE AND CHRONIC ARTERITIS, AND ORGANIC DISEASES OF THE COATS OF ARTERIES 220
Acute arteritis 220
Chronic arteritis 221
Morbid alterations in the coats of arteries, and especially the aorta 222
Causes of morbid depositions in the coats of arteries ... 226

PART III.

ORGANIC AFFECTIONS ON THE HEART AND GREAT VESSELS 232
CHAP. I. HYPERTROPHY OF THE HEART 233
SECT. I. *Anatomical Characters, with Classification and Nomenclature of Hypertrophy* 233
Classification and Nomenclature 234

Page
Natural dimensions and weight of the heart ... 235
Anatomical characters of hypertrophy 237
SECT. II. *Mode of formation, with the predisposing and exciting causes of Hypertrophy* 242
Mode of formation and predisposing causes 242
Exciting causes 245
SECT. III. *Order of Succession in which the several Compartments of the Heart are rendered Hypertrophous by an obstacle before them in the course of the Circulation* 248
Comparative frequency of the forms and combinations of hypertrophy 251
SECT. IV. *Pathological effects of Hypertrophy* 252
Effects of hypertrophy of the left ventricle, and of organic diseases of the heart in general, on the brain ... 255
SECT. V. *Signs and Diagnosis of Hypertrophy* 262
General signs 263
1. Palpitation 263
2. Dyspnœa 264
3. Cough 265
4. Hæmoptysis 265
5. Pulse 265
6. Affections of the head 266
7. Complexion 267
8. Serous infiltration 267
9. Angina cordis 268
General signs of hypertrophy of the right ventricle ... 268
General signs of hypertrophy of the auricles 270
Physical signs—Impulse 270
Sounds 274
Dulness on percussion 276
Prominence of the præcordial region ... 276
SECT. VI. *Progress, Terminations, and Prognosis of Hypertrophy* ... 277
Progress and Termination 277
Prognosis 281
SECT. VII. *Treatment of Hypertrophy* 281
Appendix to hypertrophy 291
CHAP. II. DILATATION OF THE HEART 293
SECT. I. *Anatomical characters with Classification and Nomenclature of Dilatation* 293
SECT. II. *Mode of formation, with predisposing and exciting Causes of Dilatation* 297
SECT. III. *Pathological effects of Dilatation and Mode of their Production* 299
SECT. IV. *Signs and Diagnosis of Dilatation* 305
General signs 305
1. Serous infiltration 306
2. Discolouration of the face 306
3. Congestion of the brain 307
4. Injection of the mucous membranes 307
5. Passive hæmorrhage 307

Page
6. Congestion and enlargement of the liver ... 307
7. Angina of the heart 308
General signs of dilatation of the right ventricle ... 308
General signs of dilatation of the auricles 309
Physical signs 309
Impulse 310
Sounds 310
Resonance on percussion 314
Physical signs of dilatation of the auricles 315
SECT. V. *Progress, Terminations, and Prognosis of Dilatation* ... 315
Prognosis 316
SECT. VI. *Treatment of Dilatation* 316
CHAP. III. PARTIAL DILATATION OR REAL ANEURISM OF THE HEART ... 320
Abstract of Mr. Thurnam's paper 321
Aneurism of the left ventricle 321
Aneurism of the auricles 331
CHAP. IV. SOFTENING OF THE HEART 332
Anatomical characters of softening 332
1. Red softening 333
2. Whitish softening 335
3. Yellow softening 336
Signs and diagnosis of softening 338
General signs 338
Physical signs 339
Diagnosis 339
Prognosis 340
Treatment 340
Cases of softening 341
CHAP. V. INDURATION OF THE HEART 346
CHAP. VI. ADIPOSE AND GREASY DEGENERATIONS OF THE HEART ... 347
Excess of fat 347
Greasy degeneration 348
Atrophy and œdema of the adipose tissue 348
Cases of fatty heart, illustrating the signs 348
CHAP. VII. OSSEOUS, CARTILAGINOUS, AND OTHER ACCIDENTAL PRODUCTIONS CONNECTED WITH THE MUSCULAR SUBSTANCE OF THE HEART, AND WITH THE PERICARDIUM 353
CHAP. VIII. ATROPHY OF THE HEART 356
CHAP. IX. DISEASES OF THE VALVES AND ORIFICES OF THE HEART ... 358
SECT. I. *Anatomical Characters, with predisposing and exciting causes, of Diseases of the Valves* 358
Induration of the mitral valve 362
Induration of the aortic valves 365
Induration of the valves at the right side of the heart ... 366
Predisposing causes of valvular disease 367
Exciting causes 367
SECT. II. *Anatomical characters and causes of Warty Vegetations of the Valves* 368
SECT. III. *Pathological effects of Disease of the Valves, and mode of their Production* 371

Page
SECT. IV. *Signs, Diagnosis, Prognosis, and Terminations of Disease of the Valves* 374
General signs of disease of the valves 374
Pulse in valvular disease 375
—— in disease of the mitral valve 376
—— in contraction of the aortic valves 378
—— in regurgitation through the aortic valves ... 379
—— in valvular disease of the right side 380
Pain in valvular disease 381
Progress, Terminations, and Prognosis 381
Physical signs 382
Signs of disease of the aortic valves 383
——————— of the pulmonic valves 385
——————— of the mitral valve 387
——————— of the tricuspid valve 388
——————— of the arterial and auricular valves conjointly 388
Diagnosis of valvular from inorganic murmurs ... 389
Unusual and curious sources of murmurs 391
SECT. V. *Cardiac asthma* 393
General doctrine of asthma 394
1. Asthma from chronic dry catarrh 395
2. Asthma from pituitary catarrh 395
3. Asthma from mucous catarrh 395
4. Asthma from disease of the heart 396
5. Asthma from spasmodic constriction of the bronchial tubes 397
Cardiac asthma described 398
Asthmatic fit described 401
SECT. VI. *Treatment of Valvular Disease* 405
Bloodletting 407
Diuretics 408
Purgatives 409
Diaphoretics 410
Emetics 410
Puncturing 411
Setons, Issues and Blisters 412
Expectorants 412
Gases 413
Antispasmodics 413
Stomachics 415
Tonics 416
Diet 416
CHAP. X. ANEURISM OF THE AORTA 418
SECT. I. *Classification, Nomenclature, Anatomical Characters, and Formation of Aneurism of the Aorta* 418
Classification 418
1. Dilatation, or enlargement of the whole circumference of the aorta 419
2. True Aneurism, or lateral, partial, dilatation of the aorta 421

Page
3. False aneurism, or aneurism by ulceration of the internal and middle coats 422
4. Mixed aneurism, or false aneurism surmounting true ... 425
General observations on aneurism of the aorta 425
SECT. II. *Pathological Effects of Aneurism of the Aorta on Contiguous Parts* 428
1. Compression of contiguous parts 429
2. Destruction of contiguous parts 429
SECT. III. *Signs and Diagnosis of Aneurism of the Aorta* ... 434
General signs 435
Physical signs 441
Sounds 442
Purring tremor 446
Pulsation 447
Case of aneurism immediately behind the heart ... 447
Strong and double jogging impulse from this cause ... 449
SECT. IV. *Synopsis of the Physical, in conjunction with the General Signs, in reference to the several varieties of Aneurism of the Aorta* 451
1. Simple dilatation of the arch, and ascending aorta ... 451
Fallacies and methods of detecting them 451
2. Dilatation of the pulmonary artery 452
Diagnosis from dilatation and aneurism of the aorta ... 453
3. Sacculated aneurism of the thoracic aorta 453
Fallacies, and methods of detecting them 455
4. Sacculated aneurism of the abdominal aorta 457
Fallacies and methods of detecting them 459
Case of aneurism of the abdominal aorta 463
Anæmic and Nervous Pulsation of the Abdominal Aorta ... 464
Its physical signs 464
Its general signs 465
Aortic Pulsation from Enteric Inflammation 465
APPENDIX TO ANEURISM OF THE AORTA 466
Case of aneurismal pouch of the aorta bursting into the right ventricle 466
Case of rupture of a dilated aorta into the pulmonary artery 469
Signs of aneurism of the origin of the aorta opening into the right ventricle 471
Signs of aneurism of the aorta opening into the pulmonary artery 472
Diagnosis of aneurism of the aorta opening into the right ventricle or the pulmonary artery from other diseases ... 472
SECT. V. *Spontaneous Cure and Medical Treatment of Aneurism of the Aorta, and Treatment of Nervous Pulsation* ... 473
Spontaneous cure 473
Medical treatment 474
Treatment of anæmic and nervous pulsation 481
CHAP. XI. MALFORMATIONS OF THE HEART 482
Communication between the two sides of the heart 484
General signs of communication between the sides of the heart 487

Page
Physical signs 489
Cases of communication between the two sides of the heart ... 491

PART IV.

NERVOUS AFFECTIONS OF THE HEART ... 495
Spasms, Convulsions, and Paralysis 495
CHAP. I. NEURALGIA OF THE HEART, OR ANGINA PECTORIS 496
Causes of angina pectoris 497
Nerves affected in angina 499
Diagnosis 500
Treatment 500
CHAP. II. PALPITATION, PARTICULARLY NERVOUS 503
Palpitation from inorganic causes, usually called nervous, imitating disease of the heart 504
1. From dyspepsia, hysteria, &c. 505
2. From Anæmia 507
General symptoms of anæmia 508
Physical signs 509
Treatment of anæmia 510
3. From too stimulant diet 510
4. From plethora 511
Cases illustrative of palpitation 512
Case 1. Dilatation with hypertrophy; overfeeding; simple apoplexy 512
Case 2. Dyspeptic and nervous palpitation, with dilatation and cerebral congestion 513
Case 3. Dyspeptic, hypochondriacal, and nervous palpitation ... 514
Case 4. Plethoric dyspepsia, with palpitation 515
Case 5. Plethoric congestion of the heart, with palpitation and slight dyspepsia 516
Case 6. Plethora; dyspepsia; hepatic enlargement; jaundice; intermission; palpitation; "fulness" of the heart; and fainting ... 517
Case 7. Plethora; bilious engorgement; intermission of the pulse; occasional fainting; great oppression and debility ... 520
Case 8. Derangement of the stomach, bowels, and liver; paroxysm of palpitation with orthopnœa 521
CHAP. III. SYNCOPE 522
Treatment 523

PART V.

MISCELLANEOUS AFFECTIONS 524
CHAP. I. POLYPUS OF THE HEART 524
Anatomical characters 525
1. Unorganized polypi 526

Page
2. Slightly organized polypi 526
3. More completely organized polypi 528
Causes and formation of polypous concretions 528
1. From mechanical retardation of the circulation 528
2. From inflammation 529
Signs and diagnosis of polypi 530
Physical signs 531
General signs 531
Treatment 532
Solubility of polypous concretions 535
CHAP. II. DISPLACEMENTS OF THE HEART 536
Symptoms 537
Diagnosis 537
CHAP. III. HYDROPERICARDIUM 538
Signs and diagnosis 538
Treatment 539
CHAP. IV. PNEUMOPERICARDIUM 540

PART VI.

Cases 542
Description of the plates 613
Plates (Figs. 2 to 22, inclusive) 622
Table of pulses of disease of the heart 623
Appendix of the autopsies of V . . . ., Esq., and Goff ... 625
Alphabetical index 627

INTRODUCTION

TO THE FIRST EDITION.

Doubts of the profession respecting the applicability of auscultation to the diseases of the heart, xxii. *These doubts not without foundation,* xxiii. *Sketch of the deficiencies left by preceding writers:* viz. *Incorrect view of the action of the heart by Laennec,* xxiii. *Error of attributing the same symptoms to all its diseases indiscriminately,* xxiii. *This error partly corrected by M. M. Bertin and Bouillaud, but their generalizations not perfectly accurate,* xxiv. *Errors in auscultation resulting from the incorrect view of the heart's action, by Laennec,* xxv. *Murmurs and purring tremors of the heart and arteries independent of organic disease, attributed by Laennec to a wrong cause,* xxv. *The more accurate doctrines contained in his first edition retracted in his second,* xxv. *Murmurs and purring tremor, from whatever cause, attributed by the writer to modifications in the motion of the blood,* xxvi. *Diagnosis facilitated by this view,* xxvi. *Researches of preceding Authors on the signs of Aneurism of the Aorta, pericarditis, and polypi in the heart, inconclusive,* xxvii. *Treatment of diseases of the heart open for improvement,* xxvii. *Neglected by Laennec,* xxvii. *Too cursorily treated by Bertin and Bouillaud,* xxvii. *Principles of the latter not always perfectly sound,* xxvii. *Writer has therefore attended particularly to the treatment,* xxviii. *Reply to those who despair of improvement in treatment,* xxviii. *Direct improvements described,* xxviii. *Collateral* *improvements in reference to apoplexy*, xxix. *To disease of the heart imitating dyspepsia*, xxix. *To dyspeptic, anæmic, and nervous affections imitating disease of the heart*, xxx. *To asthma and dropsy*, xxx. *To rheumatism with pericarditis*, xxx. *To enlargement of the liver and abdominal dropsy*, xxxi. *To all febrile and inflammatory affections connected with disease of the heart*, xxxi. *Treachery of the pulse*, xxxi. *Opinion of Senac as to the importance of studying diseases of the heart*, xxxii. *Plan of the work*, xxxii. *Symptoms and morbid anatomy studied as cause and effect*, xxxiii. *Mode in which the cases were collected*, xxxiii. *At what hospitals*, xxxiv. *Intentional repetitions adverted to*, xxxiv. *Superfluous minutiæ suppressed*, xxxiv. *Views respecting asthma*, xxxv. *Comparative value of general and physical signs*, xxxv.

PRECEDED by names so distinguished as those of Corvisart, Kreysig, Burns, Laennec, and Bertin, I am sensible that I expose myself to the imputation of presumption, in offering to the profession a new treatise on the diseases of the heart and great vessels. I feel called upon, therefore, to explain, in a more circumstantial manner than I should otherwise have wished, the motives which have induced me to undertake this work, and the plan which I have pursued in its execution. Whether I am justified in the attempt I can scarcely form an opinion. Every author contrives, I believe, to persuade himself that the work which consumes his own midnight oil, is precisely the one that is wanted. It is for the reader to determine whether I labour under the delusion common to my brethren.

Notwithstanding the strong light diffused over the diseases of the heart by the researches of the above-mentioned authors,—notwithstanding the brilliant sunshine emanating from the discovery of auscultation by Laennec,—a discovery, which, according to M. Bertin, "has, in a few years, more completely illumined the diagnosis of the diseases in question, than all the other modes of exploration had done for two centuries;" the great body of the profession still deny that the piercing ray has reached its destination, still doubt the utility of auscultation in reference to the primary organ of the circulation, still find the ordinary symptoms beset with their accustomed difficulties, still complain, in short, that the obscurity which involves the diseases of which we speak, is scarcely less profound than ever :* and, while conflicting opinions are embarrassing the judgment, and undermining the confidence of the patient investigator of truth, there is a general outcry for an additional mass of well-attested evidence, which may bring the subject to some kind of a conclusion.

It rarely happens that a general impression is wholly unfounded: nor is it, if I mistake not, in the present instance. Authors actually have not succeeded in completely redeeming this subject from its obscurity. Errors remained to be corrected, deficiencies to be supplied, inconsistencies to be reconciled: the subject — a confused and incongruous mass, required to be moulded and compacted into a symmetrical and harmonious whole, the parts of which, while perfect in themselves, should, by their justness of proportion and unity of design, afford relief and support to each other.

I proceed to glance briefly at the subjects where the principal defects appear to have resided; and this I do, not only for the purpose of general guidance to the student, but also for that of pointing out where I have differed from preceding writers.† In these differences, I am anxious to offer my opinions, not as established facts, though I trust that they will be found grounded on careful observation, but simply as propositions to be admitted or rejected according to the test of general experience. I am satisfied that, in our profession more especially, where there are few *fixed* points to constitute the basis of an inductive process, nothing is more difficult to ascertain than a *general* fact. Innovations, therefore, cannot be regarded with too much suspicion, cannot be scrutinised with too much severity, cannot be received with too much caution and reserve.

The most prominent error which reigns throughout the doctrines of Laennec, and which has prevailed in the schools since the first publication of his work, is, that he mistook the nature of the action of the heart. I trust that the view which I have ventured to substitute may be found more satisfactory; and, as nearly a year and a half has elapsed since I first published my experiments and clinical observations relative to it: as my conclusions have, throughout that period, remained, so far as I can judge, uninvalidated; and as I have recently repeated the experiments with the same results, before a number of the most distinguished physiologists and pathologists of the metropolis;* I hope I shall not be considered precipitate in having taken the decisive step of modifying and explaining all the physical signs of disease of the heart according to the view in question.

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* A distinguished Frenchman recently said to me, " Monsieur, je ne crois pas, pour vous dire la vérité, que l'on puisse en faire le diagnostic—que sur la table du salon."

† The further defects, which have been supplied in the present edition, are briefly enumerated in the preface.

Laennec and his predecessors have assigned to diseases of the heart a certain series of symptoms, which they conceived to be common to the whole; but they had not analysed those symptoms, and ascertained which were peculiar to, and pathognomic of, the several affections taken individually. M.M. Bertin and Bouillaud, both writers of high talent, made this attempt, and with partial success; but the spirit of generalization (if I am correct in my own views) carried them a grade too far. What observation leads me to regard as an inaccuracy, constitutes the hinge of their work—the pivot on which turns the principal train of their reasoning: namely, that the symptoms of a retarded circulation are, under all circumstances, the result of a *mechanical obstacle* to the course of the blood:—that when, for instance, they accompany hypertrophy or dilatation, they are not consequences of these affections, but of some co-existent mechanical obstacle, as a contracted valve, an aortic aneurism, &c. I have attempted to show, not only that hypertrophy, dilatation, and softening can, of themselves, respectively occasion the symptoms in question; but, that these symptoms are seldom produced in any very remarkable degree of severity by a mechanical obstacle, unless hypertrophy, dilatation, or softening of the heart is superadded.†

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* Vid. p. 11, 13 and 20. To these may now be added, my experiments on the sounds in 1835, detailed at p. 25 et seq.

† M. Bouillaud complains that I have misrepresented his opinions in the above

It may naturally be supposed that the erroneous view which Laennec took of the heart's action, led to corresponding errors in his doctrines of auscultation. The errors are principally those of omission and of incorrect explanation. The omissions are considerable and important. He was not aware of a fact first noticed by the writer in June 1825, namely, that murmurs are produced by regurgitation through the valves. This oversight alone naturally shook the confidence of many, and eventually of himself, in his theory of valvular murmurs. For, the lesion being found in one valve, when, according to that theory, it was expected in another, the inevitable conclusion was, that the theory was incorrect. At the same time, the cause of the murmur remained doubtful.

The perplexity was further increased by the existence of murmurs independent of valvular disease, and accompanying anæmic and nervous palpitation without any organic lesion whatever. These murmurs Laennec attributed to a wrong cause: viz. to the sound of the muscular contraction, instead of to the modified motion of the fluid; which I presume to consider the true cause. Hence, he was unable to analyse and foresee the circumstances under which nervous and anæmic murmurs should occur, and, consequently, to distinguish them from those occasioned by valvular disease.

Several minor phenomena likewise, as the purring tremor, and the arterial thrill and bellows-murmur, he was, in consequence of the confusion created by the error in question, equally unable to explain. Hence, he vaguely attributed them to some unknown "modification of the nervous action."

It cannot be a subject of surprise that, with the above opinions, acquired chiefly during the latter period of his life, he should have retracted, in his second edition, the much more accurate

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paragraph. With the utmost anxiety to correct my mistake and make ample amends, I have thoroughly and carefully examined his work in 1824 and that in 1835, and I am sorry to say that I cannot detect any just foundation for his complaint. I have shown at p. 302, by quotations and references, that he really entertained the opinions which I have ascribed to him, and that he still maintains the same in his later work.

doctrines respecting murmurs as signs of valvular disease, which he had advanced in his first;—transmitting to his disciples the confusion which reigned in his own mind, but which, like the storm that, in tropic climes, is the precursor of the purest, brightest weather, must, sooner or later, had his life been spared, have rolled away before the irresistible force of his purifying and enlightening genius.

The murmurs attending valvular disease, nervous palpitation, reaction from loss of blood, and anæmic or chlorotic palpitation in general; also the allied phenomena of purring tremor, and arterial thrill, throb, and murmur,* I have attributed to modifications in the motion of the blood, and explained according to the laws of hydraulics and acoustics. In this way, not only may organic diseases of the heart be readily and certainly distinguished from nervous and other affections wearing their aspect, but, with attention to certain rules which I have offered respecting the situations where valvular sounds are to be explored, and to certain corroborations derived from general symptoms, the particular valve diseased may in general be detected with precision. Such, at least, are the conclusions to which I have been brought by a very considerable number of cases, a small proportion of which are appended to this volume.†

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* To these, the venous murmur has been added in the present edition.

† I am enabled in the present edition to speak much more decidedly even than in the above paragraph, having constructed a code of rules of so simple a nature that the particular valve presenting a murmur, whether from constriction or regurgitation, may be detected with demonstrative certainty in every instance where the murmur is distinct. In Aug. 1828, I tested these rules on four intelligent students of St. George's Hospital, professing to be total strangers to the auscultation of the heart. After employing ten minutes in giving them verbally the explanation appended to the diagrams Fig. 4. A, 4. B, 4. C, I introduced to them six patients presenting five distinct varieties of valvular disease, including the pulmonic. They delivered in writing sixteen diagnoses, of which fourteen were perfectly correct, and two only were partially defective (see Med. Gaz. Sept. 1828). I have subsequently seen numerous students and various practitioners become practical adepts in the course of a few weeks, by committing the rules and diagrams to memory and examining fifteen or twenty cases, which I constantly keep accessible in my Hospital practice. As an instance of the ease with which a student will unravel the most complex case, the reader is referred to the case of Goff, pp. 610 and 626.

The utility of particular valvular diagnosis is explained at p. 390.

The investigations of Laennec on aneurism of the aorta, were limited and inconclusive: accordingly, he remarks that, "of all the severe lesions of the thoracic organs, three alone remain without pathognomonic signs to a practitioner expert in auscultation and percussion,—namely, aneurism of the aorta, pericarditis, and polypi in the heart previous to death." I hope that my attempts to throw light on these subjects, may not be found entirely fruitless. The article on aneurism is the substance, with considerable additions, of a series of essays published in the Lond. Med. Gaz. Aug. 22, 1829, and is founded on nearly forty cases in which the diagnosis was verified by post-mortem examination. It was originally the subject of the writer's inaugural dissertation.

The treatment of diseases of the heart offers a spacious field for improvement. Previous to the discovery of auscultation, these maladies could seldom be detected before they were so far advanced as to be incurable; and then was not the time to judge of the efficacy of remedies. Laennec, absorbed in his investigation of the diagnosis, paid comparatively little attention to the treatment. His first edition scarcely alluded to it: in the second it is only cursorily treated. Bertin and Bouillaud are not more satisfactory,—giving a bold outline of leading principles, such as might be struck out by generalization in the closet, but seldom descending into those detailed delineations of therapeutic measures, which are essential to the practitioner at the bedside.

Nor are these principles always, perhaps, perfectly sound. Their habit of attributing the symptoms of a retarded circulation, under all circumstances, to one cause only,—a mechanical obstacle, gives a wrong bias to the mind; and that of entwining inflammation with the cause of almost every organic lesion of the heart or great vessels, is replete with danger to the inexperienced practitioner. While I feel bound to say this, (for it is the duty of a writer to point out the path which is insecure, no less than that which is safe,) let me not be supposed to detract from the singular merits of these authors: let me offer my tribute of admiration to the talent which shines through every page of their elegant and scientific work, and acknowledge the extensive obligations that I owe it in the execution of my own.*

Conscious of the gap that was presented in the treatment of diseases of the heart, I have devoted more attention to this, than to any other department of the subject: availing myself, in particular, of the wide and favourable sphere for observation, afforded by a long residence as House Physician and Surgeon successively, in the Royal Infirmary of Edinburgh; where living, literally, I may say, as well as figuratively, at the bed-side of the patient, I had an opportunity of closely watching every habitude and phasis of the disease—every operation and effect of remedies. The results of these researches were submitted in a memoir to the Royal Medical Society of Edinburgh, in the year 1824-5.

Many think that the expectation of effecting an improvement in the treatment of diseases of the heart, is chimerical: and they think so because, not being accustomed to recognise the diseases in question before they have attained an advanced stage, they are pre-occupied with the old and popular idea of their incurability. To such it might, perhaps, be a sufficiently philosophical answer to reply, that an improved knowledge of the nature and causes of a disease, must alone necessarily lead to an improvement in the treatment; and that therapeutic weapons are dangerous when wielded in the dark. But here we may go much farther: we may say that, by the improved means of diagnosis, the maladies under consideration may be recognised, not only in their advanced, but in their incipient stages, and even when so slight as to constitute little more than a tendency. We may say, on the grounds of incontestable experience, that, in their early stages, they are, in a large proportion of instances, susceptible of a perfect cure; and that, when not, they may, in general, be so far counteracted as not materially, and sometimes not at all, to curtail the existence of the patient. We may, accordingly, predict that the term "disease of the heart," which at present sounds like a death-knell when uttered by the physician, will hereafter become by familiarity not more alarming than the term *asthma*, under which it is frequently disguised.

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* M. Bouillaud has dwelt little more on the treatment in his Treatise in 1835, than in his previous work. For instance, he dismisses so important a subject as hypertrophy in two pages, and to dilatation he gives three lines! His treatment of the worst cases of hypertrophy by the profuse bleeding system of Albertini and Valsalva, is not only strongly objectionable for the reasons specified at p. 283, but is singularly inconsistent in a writer who has insisted so strenuously on palpitation being produced by anæmia! My equally strong objections to his extravagant, dangerous, and, after all, inefficient, and unnecessary bleedings for acute rheumatism and rheumatic inflammation of the heart, are unfolded at pp. 180 and 191, notes.

Such are the *direct* practical improvements to be expected from a better knowledge of diseases of the heart. But there are collateral ones of no less magnitude. It has been stated by M. Richeraud, repeated by Bertin, and echoed by all who are conversant with this class of maladies, that "hypertrophic enlargement of the heart is more closely allied to apoplexy and palsy than the apoplectic constitution itself.*

Should the hypertrophy be recognised, its effects on the brain may be counteracted by judicious treatment: should it be overlooked, the patient, with a view to reducing his *apoplectic fulness of habit*, is ordered smart exercise, which, by increasing the action of the heart, already too powerful, causes a preternatural determination of blood to the brain, and induces the apoplectic or paralytic seizure. According to evidence hereafter to be adduced, the majority of those who are prematurely cut off by apoplexy in the apparent enjoyment of good health, sink under the circumstances described.†

Again, there are few more common and certain exciting causes of palpitation and difficulty of breathing in disease of the heart, than derangement of the stomach. What happens to the patient in this case? Tracing the attack, in perhaps every instance, to a dyspeptic fit, he naturally concludes that the latter is the cause: that it is "all indigestion." "Good air, and plenty of exercise," are the remedies recommended: the result is an apoplectic seizure. The circumstance that before the introduction of the new mode of exploring diseases of the heart, they could rarely be detected in their early stages, contributed to the error in question. For, as patients frequently recover from the early stages, the recovery was regarded by those who assumed this class of diseases to be incurable, as a proof that the affection was merely dyspeptic. Hence dyspepsia acquired the reputation of producing certain symptoms, particularly in the head, which are in reality foreign to it, being exclusively the results of a co-existent disease of the heart.

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* This constitution consists, according to the popular idea, in a broad, robust frame, full habit, and florid complexion. It is in general attended with an unusual size and thickness of the heart.

† It is shown in the present edition, p. 256, that other diseases of the heart, besides hypertrophy, are causes of apoplexy.

There prevails another error, the converse of the above—that of mistaking anæmic, nervous, dyspeptic, and other varieties of palpitation, for disease of the heart. The frequency of cases of this kind, especially amongst men of studious habits, (and more particularly, I have noticed, among those of my own profession,) is truly surprising; and as it has always been considered difficult, and by many impossible, to distinguish the two affections, the alarm created is sometimes distressing. Having thought this subject of so much importance as to demand a separate article, (see Palpitation,) I shall here only say, that, so far as my own experience enables me to judge, the discrimination may be made with ease and certainty.

An immense proportion of asthmas—and of the most dangerous and distressing cases, result from disease of the heart: the same may be said of dropsies, especially those that are universal. If the cause be overlooked, the asthmatic is harassed with a farrago of inappropriate and unavailing, not to say pernicious remedies; and the hydropic is treated with dangerous activity, or for imaginary affections of the liver, the lungs, or the kidneys. On the other hand, if the cause be detected in the incipient stage, by precautionary measures both the one effect and the other may in general be prevented.

In acute rheumatism, there is no more common and formidable source of danger than inflammation of the heart and its investing membranes. Should it be overlooked when existing in a severe form, (and even in that form it is, to those unacquainted with auscultation, one of the most obscure and insidious of maladies,) the patient almost invariably dies from the immediate effects of the attack, or becomes a short-lived martyr to an incurable organic disease of the heart.

There is scarcely a disease of the heart, accompanied with obstruction of the circulation for any considerable period, which is not productive of enlargement of the liver, and, sooner or later, of its ordinary consequence, abdominal dropsy. Yet there are few common facts in medical science less generally known than this intimate connexion between the heart and the liver. The dropsy is ascribed to the latter; the treatment extends not beyond this organ; the unknown cause continues to reproduce its effect, and the patient, if he obtain relief at all, only obtains it to undergo a speedy relapse.*

Individuals affected with disease of the heart are peculiarly liable to inflammation of the lungs; and such inflammation, as I have endeavoured strongly to inculcate throughout this volume, is singularly rapid and destructive. Yet if, from ignorance of the state of the heart, free depletion be practised on the ordinary principles, the patient may sink suddenly after the first or second abstraction of blood. I have more than once witnessed this catastrophe, and few practitioners of experience have not seen the same.

In fever and inflammation in general, disease of the heart may impart to the pulse dangerously deceptive characters of hardness, fulness, weakness, or irregularity, and the patient may be bled too much, from the prevalence of the former characters, or too little, from the presence of the latter.†

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* Similar remarks often apply to enlargement of the spleen, to hæmorrhage from the stomach connected with congestion either of the liver or the spleen, to bleeding piles dependent on engorgement of the portal system, and occasionally even to uterine hæmorrhage.

† At p. 623 I have offered a complete table of the pulses of disease of the heart—the first, I believe, that has ever been attempted. They imitate all the pulses of ordinary disease: consequently, unless the practitioner can make allowance for disease of the heart, the pulse is a fallacious criterion of other affections. This appears to me to be

Thus it is seen that the practical improvements to be derived from a better knowledge of the diseases of the heart, extend, not to the diseases of this organ alone, but to a multitude of the most formidable maladies incident to the human frame. There is, in short, scarcely an affection with which disease of the heart may not be more or less interwoven; and "if," to use the language of Senac, "we would not pronounce rashly on an infinity of cases; if we would not harass our patients by noxious and unavailing remedies; if we would not accelerate death by treating certain diseases like others which are entirely different; nor be exposed to the disgrace of seeing our diagnosis falsified by the results of dissection; finally, if we would not have danger to be imminent, whilst we are under the blind impression that it is remote, we must study the diseases of the heart."

Such appear to be the vacuities left by preceding writers, and such the advantages to be anticipated from their being supplied. It remains for me to explain the plan of the present work, and glance at a few particulars in its execution.

The work is divided into six parts; I. The Anatomy and Physiology. II. Inflammatory affections. III. Organic affections. IV. Nervous affections. V. Miscellaneous affections. VI. Cases. Although every arrangement of diseases of the heart presents considerable difficulties, and I am by no means perfectly satisfied with the one which I have adopted, it appears to me preferable to others, because affections of the same class, being thrown together, by juxtaposition reflect light upon each other; nor, at the same time, are the inflammatory and the organic affections in general so intimately connected, as to render their separation impossible without doing violence to the continuity of the subject. The miscellaneous affections are ranged by themselves, because they are not reducible to any of the preceding heads.

In the execution of the work, it has constantly been my aim,

the main reason why there has been, from time immemorial, so much disagreement amongst authors respecting the indications of the pulse and its value as a sign of disease.

by studying the symptoms in connexion with the morbid anatomy, to trace the alliance of the two as cause and effect, and thus to reduce them to certain general and intelligible principles, which might not only contribute to future accuracy of observation, but facilitate the registration of so many and so complicated facts in the memory.

As the authenticity of cases and observations is of the first importance, I deem it necessary to present a short explanation of the manner in which I have conducted my investigations. Being persuaded that no evidence is so suspicious as that of the senses, because the magnitude of an error is in proportion to the certitude which is supposed to attach to that mode of exploration, it has constantly been my endeavour to avail myself of the collective testimony of many. Accordingly, I have, for publication, preferred hospital cases, as being the best attested; I have invariably *written* the opinions or *diagnoses* before the death of the patient; have publicly tested them by the results of post-mortem examination; have minuted the dissections with the subject before me, and according to the prevailing opinions of the individuals present; and, generally before laying down my journal, I have annexed such remark as the case suggested, while the circumstances were fresh in my recollection. Finally, I have obtained signatures where a case was very remarkable, or where there appeared a possibility of its being subsequently called in question. The cases appended to this work are nearly verbatim transcripts from journals thus kept;* and, in order that they might present a just idea of the possibility of detecting disease of the heart, I have not taken them by selection, but, excepting a few, mostly without diagnoses, have introduced the whole of which I took notes in St. George's Hospital within a definite period. They will be found, I believe, to substantiate the view which I have offered of the heart's action—according to which the physical signs are explained; and, to the practical student of auscultation, by standing in the relation of exercises to a grammar, I entertain hopes that they may prove one of the most acceptable portions of the volume.

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* Except the cases added to the present edition and dated subsequent to 1831.

The hospital researches alluded to have been conducted at the Royal Infirmary of Edinburgh, as above stated: at St. Bartholomew's, London: at La Charité, Paris, where the lessons and researches of M. M. Chomel,* Andral, and Louis afforded the most favourable opportunities for auscultation: at the Santo Spirito, Rome: and, finally, at the Marylebone Infirmary and St. George's Hospital, London. From these and private sources I have minuted a greater number of cases, than has, I believe, been published by any previous author.

In some parts, I have occasionally introduced repetitions. Thus, in describing the mode in which changes of structure produce their pathological effects, I have glanced at the symptoms; and in describing the symptoms, I have explained them, where practicable, by the changes of structure. This I have done designedly; for I am satisfied that such is the process of thought which passes through the mind at the bed side and in the post-mortem theatre; and a practical work ought to be the transcript of the mind in those two situations. I have, likewise, made occasional repetitions in the treatment with the view of saving the reader the inconvenience of frequent reference.

Wherever the subject was one of original research, or otherwise particularly important, I have been circumstantial. Aneurism of the aorta, hypertrophy, the signs of disease of the valves, &c. may be cited as instances.† Where the subject was known, I have presented those points only, of which I am myself conscious of making use in practice, suppressing many subordinate minutiæ, which, though essential to original researches, gradually become superfluous, in proportion as the alchemic process of generalization assays, and assigns their full value to leading facts.

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* I owe it to the politeness of the French nation in general, and of this gentleman in particular, to state, that he not only granted me the privilege of being one of his clinical assistants; but, as I was engaged in making drawings of morbid structure, he also allowed me the immediate use of the best specimens which his wards afforded, purposely postponing the demonstration of them to his class till the following morning.

† Also the sounds of the heart.

Accordingly, I must refer the reader to Laennec for many details, which evince the astonishing accuracy and extent of his first researches, but which are no longer requisite for practical purposes. On the subject of the morbid anatomy of the heart I have been minute,—perhaps tediously so; but it has appeared to me necessary, because there is perhaps no organ in the body, of the diseased states of which the generality are less competent judges than of the heart; and this is the source of the frequent and dangerous error of confounding organic with nervous disease, or of overlooking the former entirely.

I am prepared to expect some dissent from my views respecting asthma as symptomatic of disease of the heart. Being the results of observation, I submit them with confidence, but shall be the first to recant, should they be demonstrated to be erroneous. I learn that M. Rostan entertains similar views, but having completed my manuscript, and thinking nature a sufficient guide, I have refrained from consulting his works.

With respect to the comparative value of the general and physical signs of disease of the heart, it may be said that Laennec rather undervalued the former and over-rated the latter. This was owing principally to the general signs being less perfectly understood when he studied than they have subsequently become in consequence of being investigated with the aid of auscultation. The ardour of his early disciples, who imagined that the physical, rendered the general signs superfluous, brought auscultation into some disrepute by the inaccuracy of their diagnosis. But since the stethoscope has taken its proper place as an auxiliary only, and the diagnosis has been founded on the two classes of signs conjointly, auscultation has ranked as a discovery which will immortalize its author and form an epoch in the history of medicine.

ERRATA.

Page 119, line 13, *for* or a tone *read* on a tone
120 — 1, *for* carotid *read* jugular.
125 — 12, *for* James *read* Bowden.
230 — 10, *for* tendons *read* tendinous.
384 — 9, from bottom, *insert* 2. *after* sound.
466 — 4, *insert* artery *after* pulmonary.

PART I.

ANATOMY AND PHYSIOLOGY OF THE HEART.

CHAPTER I.

ANATOMY OF THE HEART.

As morbid anatomy and pathology are only comparative states, or the amount of a deviation from the healthy standards of anatomy and physiology, it is essential for these standards to be thoroughly understood, before the morbid deviations can be appreciated. Of the descriptive anatomy of the heart it is not, however, my intention to treat, as this subject presents no obscurity, and as it ought to be studied in much greater detail than is consistent with the plan of the present work. I pretermit, likewise, that portion of the physiology which relates to the arrangement and action of the muscular fibres, referring the reader to Stenon, Wolff, Duncan, Gerdy, and other original sources of information. It may be briefly observed, that some imagine the systole to be effected by the contraction of a certain set of fibres, and the diastole by that of another; that, in short, the latter, as well as the former, is the result of an *active* muscular effort. This, however, has not yet been satisfactorily demonstrated; and, while awaiting the issue of further research, it is perhaps safer, for the present, to attribute the diastole to that power by which a muscle reverts from the state of contraction to that of relaxation, and which I shall, for the sake of avoiding circumlocution, designate by the title of *elasticity*.

There is one point, which is generally treated in too cursory a manner by descriptive anatomists, and the thorough knowledge of which is absolutely essential to the study of diseases of the heart. I allude to the relative size of the organ to the whole frame, and of its several compartments to each other. It is ignorance in this respect that has for centuries caused thickening, attenuation, enlargement, and diminution to be overlooked, and the symptoms of disease of the heart to be attributed to any cause but the legitimate one. As the subject might escape notice if introduced in this place, I have treated of it immediately before describing the anatomical characters of hypertrophy, where it will be both conspicuous and convenient. The weights and measurements of M. Bouillaud are also added.

A knowledge of the exact situation of the heart is a point of no less importance to the auscultator; and, though it does not strictly come under the head of anatomy, I shall, for convenience, advert to it here. The drawing opposite to the title-page illustrates the following description.

As the apex and body of the heart are free, while the base, secured by the great vessels, is comparatively, though not absolutely fixed, the organ turns in a slight degree upon its base with each alternate movement of the diaphragm, the descent of the muscle causing its longitudinal axis to assume a more vertical position, and the ascent throwing it transversely to the left. It is necessary, therefore, that the auscultator fix upon some given point at the base, which may serve as a mark and guide for his exploration of the situation of the organ. The point which to myself has appeared the most certain, is the pulmonary artery. This vessel, near the place where it divaricates into the two trunks distributed to the lungs, bulges, while the subject is horizontal, at the interspace between the second and third left ribs close to the sternum—a circumstance which, as well as the situation of the other parts of the heart, I have carefully ascertained by forcing needles through the thoracic walls, at given points, into the viscera beneath. The situation of the pulmonary artery was also well displayed by the dilatation of that vessel described in the case of Weatherly. At the spot alluded to, namely, between the second and third left ribs, close to the sternum, the second sound of the heart is louder even than opposite to the pulmonic valves themselves. This is simply because the sternum is not interposed; for the sound attains its maximum of intensity when the subject lies inclined to his left side, by which the pulmonary artery is forced as far as possible beyond the outline of the sternum; and, on the contrary, when he lies inclined towards his right side, by which the vessel is drawn under the sternum, the sound is no longer peculiarly audible between the second and third ribs. I have derived a further confirmation of the same fact from observations made on a patient shown to me by Mr. Mayo, and subsequently on three others, in whom pleuritic effusion in the left cavity of the chest had protruded the heart to the right side of the sternum (See Displacements). Here the sound of the aortic valves was as loud between the second and third ribs *on the right side*, as that of the pulmonic valves naturally is in the corresponding situation on the left. When the patient is in the erect position, the gravitation of the heart straightens and pulls down the pulmonary artery, so that the sound is less audible in the second costal interspace.

A line drawn from the inferior margins of the third ribs across the sternum, passes over the pulmonic valves a little to the left of the mesial line, and those of the aorta are behind them, but about half an inch lower down. From this point the aorta and pulmonary artery ascend; the former inclining slightly to the right, coming in contact with the sternum when it emerges from beneath the pulmonary artery, and following, or perhaps rather exceeding, the mesial line, till it forms its arch; the pulmonary artery, which is, from the first, in contact with the sternum, inclining more considerably to the left, until it arrives at the interspace between the second and third ribs above described. A vertical line, coinciding with the left margin of the sternum, has about one third of the heart, consisting of the upper portion of the right ventricle, on its right; and two thirds, composed of the lower portion of the right ventricle and the whole of the left, on its left. The apex beats between the cartilages of the fifth and sixth left ribs, at a point about two inches below the nipple, and one inch on its sternal side.

The lungs descend along the margins of the sternum about two inches apart, and overlap the base of the heart, slightly on the right side, and more extensively on the left: then, receding from each other, they leave a considerable portion of the right ventricle, and a less extent of the lower part of the left, in immediate contact with the thoracic walls.

The right auricle is in front of the heart, at its right side and upper part. One portion of it is overlapped by the right lung, and another, principally the appendix, is in contact with the sternum. The left auricle is situated deeply behind and to the left of the heart at its upper part, opposite to the interval between the cartilages of the third and fourth ribs. The extremity of the appendix is visible in front, but, when the volume of the heart is natural, it is not in contact with the sternum, being considerably overlapped by the left lung. The auricular orifices are situated opposite to the interspace between the third and fourth ribs, and the right is rather lower down than the left. As, however, the orifices are overlapped by the lungs, the sound of their valves is much less audible immediately over them than near the apex of the heart, to which part the sound is conducted by the chordæ tendineæ and columnæ carneæ. The pericardium ascends on the great vessels as high as the commencement of the arch of the aorta, and opposite to the second ribs.

When the heart is enlarged, its longitudinal axis becomes placed more transversely, and its lateral diameter is increased. Hence, the right ventricle projects more considerably to the right, sometimes under the whole breadth of the sternum; and the left extends far beyond its usual limits to the left, sometimes elevating by compression that portion of the lung which overlaps it, so as to bring nearly its whole surface, and the tip of the auricular appendix, into contact with the walls of the chest. In addition to being broader and placed more transversely, the organ descends lower than natural—its apex sometimes beating between the sixth and seventh ribs, and its pulsation extending to the epigastrium.

When the right auricle is dilated or gorged, it extends upwards and to the right, and comes more extensively in contact with the sternum.

When the pericardium is distended to the utmost with fluid, it forms a pear-shaped bag, the top or narrow extremity of which, when the patient is horizontal, sometimes mounts even above the second rib: its sides are nearly in contact with the sides of the heart, while its front is separated from the anterior surface of the heart, in the dead subject horizontally placed, by two or three inches of interposed fluid.

From the above description, the auscultator will understand in what situations to explore the lesions of the various parts of the heart. In the section on *Murmurs from Valvular Disease*, it will be shown that this knowledge is rendered available to the detection of the individual valvular diseases, by a process so simple as to divest the diagnosis of almost all difficulty.

The situation of the heart with respect to the exterior is influenced by a few other circumstances which remain to be specified.

The heart is, by its own gravitation, withdrawn, in some degree, from the anterior walls of the chest when the subject leans or lies back, especially if inclined a little towards the right side. The same effect is produced by full inspiration, even in the erect position. Under these circumstances, as the apex touches the walls by a smaller point of contact, the impulse is weaker; and, as an increased thickness of lung, a bad conductor of sound, is interposed, the first sound is duller. On the contrary, when the subject leans forward and a little to the left, the heart, displacing the lung by its gravitation, comes into more than ordinarily extensive contact with the walls of the chest. The same effect is produced by full expiration, even in the erect position. Under these circumstances the impulse is stronger, and the first sound louder. The auscultator will know how to avail himself of these facts in the exploration of disease, and will make due allowances in his estimate of the degree of impulse and sound.

The percussion of the organ is so intimately connected with its anatomical relations, that I may perhaps be allowed to sacrifice strictness of arrangement to practical convenience, and advert to the subject at present.

Percussion on the back of one or two fingers, *firmly applied* to the chest, either on the ribs which is better, or on the intercostal spaces if necessary, is sufficiently delicate for all practical purposes, and is so convenient that I have, for seven or eight years, adopted this, to the exclusion of all other modes of mediate percussion. I must, however, admit, both from having attended the original experimental researches of M. Piorri, and from considerable subsequent experience, that the *plessimeter* invented by that gentlemen, when lined with wash-leather or cloth to prevent its clacking, is an instrument of perhaps superior nicety, in the hands of one well accustomed to it.

It is scarcely necessary to say that percussion over a solid, as the heart where it is in contact with the walls of the chest, elicits a dead sound; while that over a body containing air, as the lungs, stomach, &c., produces a hollow sound. It is less generally known, that a solid beneath a body containing air, as the liver beneath the edge of the lung, the outline of the heart beneath the lung that overlaps it, &c., may be recognised by a sound intermediate between hollow and dead. Had M. Piorri, to whom this discovery is due, explained the principle of the phenomenon according to the laws of acoustics, he would at once have made it obvious that what has often been regarded as the offspring of his own imagination, was the necessary consequence of an immutable law of nature. Thus, when sonorous vibrations of the air impinge on a non-resonant or inelastic surface, as drapery, they are arrested and the sound becomes deadened. The pedal and damper of a piano are constructed on the same principle, the only difference being, that the check is given to the vibrations of the wires themselves, instead of to those of the air. Thus, when a note is struck, the vibrations continue till the finger is raised from the key, simultaneously with which action the damper falls on the wires, and by arresting their movement suspends the sound. By depression of the open pedal, the damper is permanently raised, and the vibrations then continue, whether the finger be removed from the key or not. To apply this principle to percussion of the chest,—sonorous vibrations excited in the lung are arrested when they impinge upon a solid, inelastic body beneath, as the liver, heart, &c.; hence the sound is speedily deadened or flattened; in other words, the resonance is of a dull, flat character: whereas, when there is no subjacent solid body, the sonorous vibrations expand freely, and yield a proportionably hollow sound. To elicit these characters distinctly, a loud sound should be produced by strong percussion, and by pressing the fingers or plessimeter firmly down, so as to condense the soft wall of the chest, and render it a better conductor of sound. Having just tried the experiment before several individuals placed at remote parts of a spacious room, I find that they readily distinguish the full, hollow tone of the middle lobe of the lung, the duller intonation of the lung overlapping the heart or liver, and the dead sound of the præcordial region where the heart is in contact with the walls of the chest.

Now, it is the object of cardiac percussion to ascertain the extent of this dead sound or dulness; because, as observation has demonstrated that it increases in proportion to the increased volume of the heart, and *vice versâ*, it indicates with considerable nicety the actual dimensions of the organ. The mode of manipulation which I employ, is to place one finger over the decidedly dead part, and another over the slightly resonant edge of the lung, when, by striking the two fingers alternately, the arched line along which the organ lies in contact with the walls, may be traced with surprising accuracy, unless the subject be remarkable for obesity, which obscures the resonance. In females, the mamma may be pushed upwards, which generally leaves the dull portion sufficiently accessible.

The extent of the dull portion in an average-sized adult with a well-proportioned heart, is represented by a circle of one and a half to two inches in diameter, supposing the individual to stand or lie without protruding the chest, and in a middling state of inspiration.

When the heart is enlarged, as by hypertrophy, dilatation, fat, or even temporarily by congestion, the descent of the lungs being impeded, the dull portion increases and may attain the diameter of three, four, or, in extreme enlargement, even five inches. The centre of the dulness generally lies between the cartilages of the 5th and 6th ribs, but in great enlargement it lies lower, as between the 6th and 7th ribs, because the organ is depressed by its own gravitation, except when held up by adhesion of the pericardium. In copious hydropericardium, I have known the dulness ascend under the sternum, in the conical form of the sac, as high as the level of the second rib; and I have repeatedly traced the gradual descent of the dulness in proportion as the fluid was absorbed.

The causes which may prevent the development of dulness on percussion are, 1. emphysema of the lungs, occasioning their protrusion in front of the heart; 2. the chicken-breasted conformation of the chest, especially when connected with spinal gibbosity. Of the latter cause, though not mentioned by authors, I have noticed many instances.

CHAPTER II.

ON THE ACTION AND SOUNDS OF THE HEART.

SECTION I.

EXPERIMENTAL RESEARCHES ON THE ACTION OF THE HEART.

When the ear or a stethoscope is applied to the præcordial region, two successive sounds, followed by an interval of silence or repose, are distinctly heard. The first, which is synchronous with the impulse, and, in vessels near the heart, with the pulse, is duller and longer, very like that produced by jerking a cord as thick as a swan-quill; the second is shorter, clearer, and smarter, like the flap or click of a bellows-valve, or it is still more closely imitated by lightly tapping the tense knuckle of one hand held close to the ear, with the soft end of a finger of the other. These sounds, though, according to Raciborski, not wholly unknown to Galen, Harvey, Haller, Senac and Corvisart, were first brought into notice by Laennec, and were attributed by him, the one to the ventricular, the other to the auricular contraction. His doctrine remained unquestioned for a period of eight or ten years, until Mr. Turner, supported by the authority of the old physiologists, Haller, Harvey, Lancisi, &c., pointed out that the auricular contraction, to which Laennec attributed the second sound, *preceded* the ventricular, and, consequently, that his theory was erroneous. Notwithstanding the talent and ingenuity displayed by Mr. Turner in proving this, he was not equally successful in assigning the cause of the second sound; and, though various theories were subsequently proposed, of which I shall give a sketch at the end of this subject, the nature of the heart's action remained a mystery, until it was made the subject of a series of experiments instituted by the writer in the summer of 1830, and repeated in that of 1831.*

From experiments on small animals, supported by analogical arguments derived from pathology, I had previously been able to infer the nature of the heart's action, almost as I subsequently found it; but the point required demonstration, and it appeared to me that the only possible mode of effecting this was, by contriving to hear the sounds at the same moment that the actions were inspected and felt: since thus alone could it be unequivocally ascertained with what motions the sounds respectively coincided. Small animals I had found insufficient for the purpose; as, in them, the sounds are too indistinct, the motions too rapid, and the impulse too feeble, to afford satisfactory data. To large animals, therefore, I turned my attention, as presenting the only means likely to lead to a solution of the question.

As many may wish to follow this investigation through all its steps, and to form their own judgment from the data presented, I insert an abstract of the whole of the original experiments, as published in the Med. Gazette, July 31, and Aug. 21, 1830: and an account of the conclusions presented by a repetition of them on August

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* These experiments were performed on the former occasion (vid. p. 15) before

Dr. Hewett, Physician to St. George's Hospital;
Mr. Smyth, House Surgeon; and
Mr. Lane, Lecturer on Anatomy to that institution;
Mr. Oswald Beale;
Mr. Frederick Julius; and
Messrs. Field, Veterinary Surgeons.

On the latter occasion (vid. p. 20) they were performed before

Mr. Babington, Surgeon to St. George's;
Dr. Burrow, Lecturer on Medical Jurisprudence to St. Bartholomew's;
Dr. Clark, Physician to the St. George's Infirmary;
Dr. Craigie, of Edinburgh;
Dr. Elliotson, Physician to St. Thomas's;
Messrs. Field, Veterinary Surgeons;
Mr. H. J. Johnson, House Surgeon to St. George's;
Mr. F. Julius, Richmond;
Mr. Mayo, Surgeon to the Middlesex Hospital;
Mr. Smyth, House-surgeon to St. George's;
Dr. Watson, Professor of Forensic Medicine to King's College, and Phys. to the Middlesex Hospital; and
Dr. Williams, Author of the "Rational Exposition of Auscultation," &c.

Mr. Brodie, who was accidentally absent, favoured me with an account of experiments by himself, which, so far as they went, coincided with my own. See his letter, p. 24.

10th, 1831. I also insert, in Section II. of this chapter, my subsequent experiments on the sounds. The young reader may, if he prefer, omit the whole, and pass on to the third chapter.

Experiments, July 31, 1830.

As, in my opinion, it is impossible to avoid fallacies when the heart is pulsating at the rate of two hundred per minute, I retarded the circulation of the rabbits which I examined, by depriving them of sensibility previous to the operation. Each pulsation was thus resolved into several distinct and successive motions, which it is philosophical to regard as an analysis of the more rapid natural action. Under these circumstances I found the auricle to contract first—not slowly—but with a motion so *rapid* as to be almost instantaneous; the moment the fluid reached the ventricle, the latter was seen to start up, evidently by the contraction of its fibres on the fluid which it contained, and not by passive distention. This was more fully proved at a later period of the experiment, when the action of the heart was from time to time suspended, and the ventricle lay quiescent, though partially *distended with blood;* for, then, the auricle often made two or three contractions, which had no stimulant effect on the ventricle; while a fourth, not more violent than the preceding, and therefore not injecting more fluid, caused it to spring up in the manner already described. Simultaneously with the springing up, commenced the retraction of the apex towards the base, by which motion the apex was thrown forward, apparently in consequence of the long axis of the heart assuming a more horizontal position. These actions constituted only the commencement of the ventricular systole: its progress was marked by a further retraction of the apex and an approximation of the sides; while the whole ventricle was elevated further forward, and its long axis rendered still more horizontal, by the auricular distention, which advanced to its maximum in the same progression as the ventricle contracted to its extreme.

On examining the posterior aspect of the heart of a frog when its action was reduced to fifteen or twenty per minute, the whole of the auricle, which had previously been concealed by the ventricle, being now exposed to view, it was found that, for a short space of time, the ventricle lay at rest *partially distended with* *blood;* the auricle then contracted with a smart brief motion—but only partially contracted, for the sinus venosus was constantly full of blood both in this experiment and those on the rabbit, and whether the circulation was quick or slow. When the auricle had relaxed again, and not till then, the ventricle (stimulated, I conjecture, by the motion, for it certainly was not by distention) was seen suddenly to rise up on its basis, to shorten its fibres, and to expel its contents, which latter action was *slowly* performed. After the completion of the systole, which was indicated by the pale colour, the diastole took place, and allowed a partial influx of blood, denoted by the return of the red colour; and in this state the ventricle remained quiescent for a short space, until again stimulated by the auricular contraction. It may be objected to this account, that as the action of the heart was preternaturally slow, the motions were anormal. We thought, however, that we could discern the same series of actions when the pulsations were forty per minute.* The Dublin Committee of the British Association for August 1835 have repeated these experiments on the frog with similar results. They correctly remark, that "in the heart of the frog, the sides of the ventricle are thin, and the cavity is large; and the increase of thickness of the sides of the ventricle caused by the contraction of its fibres, is more than counterbalanced by the diminution of volume of the ventricle attendant on the expulsion of its contents:"—in other words, the ventricle becomes very small during its systole because its walls are thin, and very large during its diastole because, from the distensibility of the thin walls, its cavity is very capacious. In this way they explain what I have frequently noticed, namely, that "during the diastole of the ventricle, its anterior surface was protuberant and approached the sternum, while its apex drooped towards the spine; and that during its systole, the anterior surface receded from the sternum, and its apex was slightly turned upwards or towards the sternum" (Exp. 4).

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* These, and various other experiments detailed in the Lond. Med. Gaz. were performed at St. George's Hospital, in the presence of a number of the medical officers and other gentlemen attached to that institution. To Mr. Babington, Surgeon to St. George's, Mr. S. Lane, Lecturer on Anatomy to the Hospital, Mr. Smith, and Mr. H. J. Johnson, House Surgeons, I am greatly indebted for their valuable aid in performing the experiments, and their patient and unbiassed scrutiny of the results.

Experiments, August 21, 1830.

I now proceed to the communication of further experiments, which, corroborated by pathological consideration hereafter to be adduced, will, I trust, be found decisive of the long controverted question respecting the cause of the motions and sounds of the heart.

At the conclusion of my experiments and researches on small animals hitherto detailed, I entertained the following impressions respecting the state of the question:—

That, in small animals, the auricular systole took place immediately *before* the ventricular, and not *after*, as supposed by Laennec, I regarded as certain, both from the evidence of my own experiments, and from the concurrent testimony of the old physiologists. It was to be presumed that the same occurred in larger animals, but it remained to be proved.

That the *impulse* and *first sound* were referable to the ventricular, and not to the auricular contraction, I was equally persuaded, 1st, because the pulse, unquestionably a result of the ventricular systole, coincided so closely, in vessels near the heart, with the impulse and sound, that these three phenomena did not admit of being ascribed to any but the same cause; 2d, because clinical observations had proved to me, that certain anormal modifications of the heart's impulse and first sound corresponded with certain morbid conditions of the ventricular, but not of the auricular parietes.

That the *second sound* did not depend on the auricular systole, was indubitable; because this preceded the ventricular contraction, whereas the sound followed it.

That it did not depend on the closure of the auriculo-ventricular valves was equally certain; because the closure of those valves takes place at the commencement of the ventricular contraction, whereas the second sound occurs after its termination. That it was not due to any other action of the auriculo-ventricular valves was obvious from physical considerations of their anatomical structure.*

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* In the first edition, November 1831, was the following passage: "That the first sound was not ascribable to the retrocession of the semilunar valves, I entertained a strong presumption, from having found the sound unimpaired, though the valves, on

Such were my impressions; but demonstrative proof was wanting, and it appeared to me that the only possible mode of obtaining it was, by contriving to hear the sound at the same moment that the action of the heart was inspected and felt; since thus only could it be unequivocally ascertained with what motions the sounds respectively coincided. Small animals were obviously insufficient for this purpose, as, in them, the sounds are too indistinct, the motions too rapid, and the impulse too feeble, to afford satisfactory data. To the larger animals, therefore, I at once turned my attention, as presenting the only means likely to lead to a solution of the difficulty.

The whole subject, then, seemed to resolve itself into the following questions, which I drew out and proposed to my coadjutors, before the operation, as the points for investigation :—

1. Do the auricles contract immediately before the ventricles?
2. Does an interval occur between the two contractions, or is the succession so rapid as to amount to continuity of action?
3. Does the ventricular contraction cause the impulse, pulse, and first sound?
4. Do the ventricles contract completely, and do they remain closed and empty, during the interval of repose? Or—
5. Do the ventricles dilate again immediately after their systole: and is this dilatation attended with an influx of blood from the auricles?
6. Is the influx of blood into the ventricles during their diastole the cause of the second sound? If not—
7. What is the cause of the second sound?

Experiment I.—An ass, of which the pulse and impulse were forty-eight per minute, was instantaneously deprived of sensation

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one side of the heart at least, were rigid with ossification; and the presumption amounted almost to certainty, from my having found the sound not only undiminished, but increased, in cases of enormous dilatation of both ventricles, in which it was impossible that the cavities could ever empty themselves; and where, consequently, the motion of the valves must have been impeded by the constant pressure of fluid on both sides (vid. for instance, case of Lambert)." This reasoning has proved incorrect; for in the subjoined experiments on the sounds, I have furnished demonstrative proof that the closure of the semilunar valves is the sole cause of the second sound. I quote the above passage to show that I was no stranger to the valvular theory—though some subsequent writers have thought that the original idea emanated from themselves.

and motion by a smart blow on the head. The trachæa was opened, a large bellows-pipe introduced, and artificial respiration maintained; while, at the same time, the left ribs were sawn through near the sternum, and forcibly bent back and broken,* so as widely and completely to expose the heart immediately behind the left shoulder: the whole was accomplished in less than five minutes.

The pericardium was next opened, and the auricles and ventricles fully displayed. The action of the heart was at first quick, tumultuous, quivering, and irregular; but after the lapse of about three or four minutes, it became regular and slower. The auricle was now seen to contract *first*, and the ventricle instantly afterwards; or, in more descriptive language, a slight contractile motion, accompanied with very inconsiderable diminution of volume, was observed to commence in the auricle, and to be propagated rapidly to the ventricle. It was not, however, so quick that it could not easily be followed by the eye; yet it seemed to be rather a continuity of action, than to consist of two consecutive parts.

The ventricular contraction appeared, and was felt by the hand to consist of a sudden energetic jerk, accompanied with a depression of the centre or body of the ventricle. This contraction was heard (through the stethoscope, applied *immediately* to the organ) to be accompanied by the ventricular sound. A note was accordingly dictated,† that, 1. *The ventricular sound was heard whilst the ventricle was seen to contract.* At an interval of time equal to that which intervenes between the first and second sounds of the heart, the contraction was followed by a sudden, jerking re-expansion or diastole, which appeared *to elevate the body of the ventricle more than the previous contraction.* Hence one of the party (Mr. Lane) expressed his opinion that it was the *diastole*, and not the systole, that occasioned the impulse. This opinion rendered it necessary instantly to repeat all our observations. The stethoscope was accordingly resumed, and several times applied by Mr. Field and the writer alternately, each counting 1, 2, synchronously with the sounds which he heard, and the impulse communicated to his ear; while others applied their hands to the ventricle, and at the same time inspected its motion. It was now proved, to the perfect satisfaction of Mr. Lane and all present, that the sound 1, and the impulse felt by the auscultator, coincided with the *visible* depression (*i. e.* contraction) of the ventricle, and the impulse felt by the hand. It was therefore dictated that, 2. *When the action of the heart was become slower, (supposed to be aboutforty per minute,) and was becoming feeble, the ventricular systolic sound and the impulse were heard, seen, and felt, both by the ear and hand, to be simultaneous.*

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* This plan was adopted in preference to cutting, in order to obviate hæmorrhage from the intercostal vessels.

† The notes were written by Mr. F. Julius to the conjoint dictation of the party during the progress, and immediately after the conclusion of each experiment; and they were finally revised and signed.

At an early part of the experiment it had been unanimously agreed that the ventricle never contracted fully, though it was then acting with great power. It was, therefore, dictated, that, 3. *The ventricular never contracted fully.*

4. *It remained apparently full during the intervals of repose* (*i. e.* from the conclusion of the diastole to the commencement of the next ventricular contraction).

On interposing the hand between the apex of the heart and the rib, which had been left above that part, the fingers were struck vigorously by the apex of the ventricle during its systole, at the moment that its body was in the act of retraction.*

As the action of the heart, after ceasing to be tumultuous, became somewhat feeble, the second sound was never very audible. It was distinctly heard, however, by Mr. Field and the writer; but as the others could not satisfactorily recognise it, a general note was deemed inadmissible, and a by-note only was dictated, the point being reserved for further investigation at the next experiment.

By-note.—*Mr. Field and Dr. Hope listened with the stethoscope alternately, and counted* 1, 2, *in unison with the sounds which they heard; while the others saw that* 1 *coincided with the ventricular systole, and* 2 *with its diastole.*

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* This is corroborated by the London Committee of the British Association repeating these experiments in August 1836. "A small opening," say they, "was made in the cartilages opposite the heart, when the stroke was perceived and felt by the fingers *inside* and *outside* the sternum at the same time, with sound, and with considerable pressure upwards against the fingers placed between the heart and the cartilages."

This first experiment was not considered conclusive. In consequence of the turbulence of the heart's action at first, and its feebleness at last, the time favourable for observation was too brief; and, consequently, a majority of the party had not complete confidence in the accuracy of their observations. This diffidence was shown by the second experiment to be greater than the case warranted.

The second experiment was performed immediately after the first.

EXPERIMENT II.—The heart of an ass was exposed to view in the same manner as before, but with still greater celerity. For *about a minute only* the action was quivering and irregular; it then fell to its natural standard (forty to fifty per minute), became *perfectly regular*, and the ventricular contraction, as felt by the hand and the stethoscope, was performed with a power which can scarcely be imagined from an examination on the outside of the chest.

Three successive motions—namely, the auricular systole, the ventricular systole, and ventricular diastole—were now distinctly recognised and acknowledged by all who witnessed them. The stethoscope was applied to the ventricle, and the *two* sounds were clearly and unequivocally heard, even by those who were unaccustomed to the instrument. Five gentlemen listened deliberately twice over, and two of them, three times, before it was dictated that, 1st. *Drs. Hewett and Hope, and Messrs. Lane, Field, and Cooper, listened successively through the stethoscope applied to the ventricle, and severally counted* 1, 2, *synchronously with the sounds which they heard; while the others ascertained, by the touch and sight, that the sound* 1 *coincided with the ventricular systole, and the sound* 2 *with its diastole.*

This part of the experiment was so deliberately performed that it occupied from ten minutes to a quarter of an hour, as near as could be judged from the whole time expended (from twenty to twenty-five minutes), and each of the experimenters was asked whether he was satisfied, whilst he had still an opportunity of renewing his examination.

It was now submitted to investigation, how the ventricular systole could occasion the impulse; since the body of the organ appeared to *recede* during that motion. The result was the following note:

2. *While the ear rested on the stethoscope applied to the middle of the ventricle, the impulse was felt by the auscultator to coincide with the systole, notwithstanding that the body of the ventricle appeared to be receding at the moment the impulse took place.*

During the course of the experiment the action of the auricle was again examined. Its anterior edge and surface only were in sight, the root and sinus being concealed behind the ventricle. It was noted that—

3. *The auricle never emptied itself, and its contraction was always very inconsiderable. The anterior edge and surface were seen to retract with a rather sudden motion; but as the extent of the motion was very inconsiderable, it had the appearance of being feeble.*

The contraction of the auricle was so much less than there was reason to anticipate from the extent of its action in smaller animals, that it was questioned whether it was, in the present instance, performed with the natural vigour. The extraordinary power with which the ventricle acted, favoured the affirmative; and as the proportion of the auricle to the ventricle is singularly less in large animals than in small, there is reason to suspect that they perform a less important function in the former.*

The inevitable conclusions deducible from these experiments are, that—

Of the Motions of the Heart—

1. The auricles contract so immediately before the ventricles, that the one motion is propagated into the other, almost as if by continuity of action; yet the motion is not so quick that it cannot readily be traced with the eye.

2. The extent of the auricular contraction is very inconsiderable, probably not amounting to one-third of its volume. Hence the quantity of blood expelled by it into the ventricle, is much less than its capacity would indicate.†

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* In subsequent experiments on *younger* and smaller asses poisoned with woorara, I found the action of the auricles greater than is here represented—especially during palpitation, where the pulse was accelerated twenty or thirty beats above its ordinary standard.

† The preceding note perhaps justifies a belief that the auricular contraction is considerable in palpitation, and is greater in young and small animals.

3. The ventricular contraction is the cause of the impulse against the side; first, because the auricular contraction is too inconsiderable to be capable of producing it; second, because the impulse occurs after the auricular contraction, and simultaneously with the ventricular, as ascertained by the sight and touch; third, because the impulse coincides so accurately with the pulse in arteries near the heart, as not to admit of being ascribed to any but the same cause.

4. It is the apex of the heart which strikes the ribs.

5. The ventricular contraction commences suddenly, but it is prolonged until an instant before the second sound.

6. The ventricles do not appear ever to empty themselves completely.

7. The systole is followed by a diastole, which is an instantaneous motion, accompanied with an influx of blood from the auricles, by which the ventricles re-expand, but the apex collapses and retires from the side.

8. After the diastole, the ventricles remain quiescent, and in a state of apparently natural fulness without distention, until again stimulated by the succeeding auricular contraction.*

Of the Sounds.

9. The *first sound* is caused by the systole of the ventricles.

10. The *second sound* is occasioned by the diastole of the ventricles.

The *immediate* causes of the sounds will presently appear in the section on the Sounds.

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* The Dublin Committee of the British Association for August 1835, have repeated these experiments and come to exactly the same conclusions: viz. 1. "In the heart of warm-blooded animals, the systole of the ventricles follows immediately the systole of the auricular appendices. 2. During the systole of the ventricles, the auricles are distended by blood from the venous trunks. 3. When their systole has ended, the ventricles become lax and flaccid; and blood passes rapidly, but not with force, from the auricles into their cavities. 4. The auricles are never emptied of their blood, and contract *but little* on their contents, an active contraction being observable only in their appendices. 6. The ventricles in their systole approach the front of the thorax, and by their contact and pressure against it, produce the impulse or beat of the heart."

Of the Rhythm.

Order of succession—

1. The auricular systole.
2. The ventricular systole, the impulse, and the pulse.
3. The ventricular diastole.
4. The interval of ventricular repose, towards the termination of which the auricular systole takes place.

Duration.

This is much the same as indicated by Laennec, viz.

The ventricular systole occupies half the time, or thereabout, of a whole beat.*

The ventricular diastole occupies one-fourth, or at most one-third.

The interval of repose occupies one-fourth, or rather less.

The auricular systole occupies the latter part of the interval of repose.

Experiments repeated, August 10, 1831.

Three asses were successively made the subject of operation, the process being conducted as before. The gentlemen present are enumerated at p. 10. It may be premised that, in consequence of the percussion of the brain not having been, in the first instance, sufficiently smart, the action of the heart was, in all three cases, more or less irregular through the greater part of the experiment, not continuing, as on the former occasions, ten or fifteen minutes almost without the slightest intermission. Notwithstanding, as the action was maintained for an equal, if not longer time, the periods of regular pulsation were sufficiently numerous and prolonged to allow of deliberate examination. The irregularity led to one important discovery which had hitherto escaped me; namely, *that the movements of the ventricles with their corresponding sounds continued perfect while the auricles were motionless.*

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* Mr. Bryan, however, performed the following ingenious experiment with a different result. He caused a long tape to pass at a pretty uniform velocity across a table, and dotted it with ink in a hair pencil synchronously with each sound of the heart heard through a stethoscope. He found that eleven inches of the tape passed on from the time of the commencement of the first sound to that of the second, and that twenty-seven inches more passed before the next sound—thirty-eight inches passing during the time of a whole beat. Thus the duration of the ventricular systole is less than one-third of that of the whole beat.—*Lancet, Jan.* 12, 1833.

The following queries were circulated to the individuals present a few days previous to the experiments. They were severally read after each of the three experiments, and the answers were the conjoint dictation of the party, partly during the experiments, and partly at the successive recapitulations.

1. *Do the ventricular systole, the first sound, the impulse, and the pulse coincide?*

A. They coincide perfectly, except that sometimes there appeared to be a *barely* appreciable interval between the impulse or first motion of the ventricle (as seen, and also felt with the fingers interposed between the apex and the ribs) and the pulse in the radial artery under the shoulder.

Remark.—The interval alluded to was ascribed to the distance of the artery from the heart. In more remote arteries it is proportionably greater, and in those near the heart it does not exist at all.

This subject had, I believe, been examined experimentally by Dr. Stokes and Mr. Hart, of which I was not aware. The fact is now well ascertained. The Dublin Committee of the British Association, Aug. 1835, have illustrated it by a very pretty experiment on a calf.

" A tube having been introduced through a puncture in the left ventricle, and one of the mesenteric arteries having been exposed and opened, the jet from the ventricle was observed to precede the jet from the arteries, by an interval easily appreciable. The femoral artery was opened, and a similar observation was made." (Exp. 1.)

2. *Do the ventricles expel the whole, or a part only, of their contents; and what is their state during the interval of repose? Are they full or empty?*

A. The ventricles not being transparent, it is not demonstrable whether they expel the whole of their contents; but the diminution of their volume by the systole is not in general so great as to convey that impression. During the interval of repose they are full, being restored to that state by the diastole. By *fulness*, is not meant *distention*, this being an ulterior degree.

Remark.—The question whether the ventricles expelled the whole of their contents or not, originated in an opinion, which had been maintained, that they did so, and, by the collision of their internal surfaces, occasioned the second sound. As this sound is proved to result from the diastole, the question becomes redundant, and its determination unimportant.

3. *With what motion of what part does the second sound coincide, and what is its cause? Is it the ventricular diastole?*

A. The second sound coincided with a motion, sensible to the touch and sight, by which the ventricle returned from its systole to the same state, with respect to size, form, and position, as before the systole. This motion was the relaxation or diastole.

4. *Do the auricles contract before, or after the ventricles, with respect to the interval of repose?*

A. Evidently before, being instantly followed by the ventricular systole. The interval of repose distinctly falls between the ventricular diastole and the auricular systole, the repose of the ventricles continuing through the auricular systole to the next ventricular systole. Such were the phenomena observed during the short periods when the motions of the auricles were regular; but, for the most part, there was either no perceptible motion in them, or the motions were irregular and bore no relation whatever to the ventricular movements.

Remark.—From subsequent experiments on rabbits, in the performance of which I was favoured with the assistance of Dr. Hewett, and Mr. Daniel, Surgeon, of Ramsgate, I am led to believe that the irregularity of the heart's action is an incidental circumstance, dependent on the mode in which the animal is stupified, and artificial respiration maintained: consequently, that it is capable of being obviated. At the suggestion of Sir B. Brodie, I stupified the rabbits in question by inoculating them with woorara poison. In the first experiment, after the expiration of a few minutes, stupefaction took place so suddenly that the action of the heart was irrecoverably extinct before artificial respiration could be established. In a second instance, arrangements having been made to establish it more expeditiously, the action of the heart was maintained in the greatest perfection, after the cerebral life of the animal had become completely extinct. We now found that, on temporarily suspending the respiration, the heart instantly became gorged, of a black colour, and distended to nearly double its natural size, while its motions were either an irregular, occasional flutter, or were wholly suspended. On resuming the inflation, the motions gradually became more and more frequent, extensive, and regular, while the distention and blackness decreased in the same proportion; until, at length, the organ regained its previous colour and dimensions, and beat with its accustomed energy and regularity at the rate of 200 per minute. This process was repeated again and again for nearly an hour; and more than once, the action was renovated, though with difficulty, after both the ventricles and auricles had rested some seconds in a state of complete immobility. Hence it appears that, when the stupefaction is complete, (as it is from woorara poison,) and artificial respiration is adequately maintained, the action of the heart may be kept regular: and it was from greater success in these two circumstances that, in my first experiments on asses, the regularity was so remarkable. The hammer employed had a smaller head, its surface, which was slightly excavated, not exceeding an inch in diameter. By this, a corresponding portion of the skull was depressed, whence the extinction of cerebral life was instantaneous and complete, and thus the performance of artificial respiration was rendered more easy. I mention these particulars, in order that, should it be found necessary to repeat the experiments, an unnecessary destruction of life may be avoided. I may add, that the experiments on the rabbit afforded an instructive exemplification of the manner in which congestion of the heart takes place in excessive dyspnœa, in suffocation, and in the agony of death. They also showed how, under these circumstances, both the impulse and sounds, even of the most enlarged heart, may be diminished or become totally extinct. To return from this digression—

5. *Do the auricles contract slightly or extensively?*

A. Very slightly, and principally at their appendix, the motion running vermicularly into the ventricular systole.

Remark.—When several irregular ventricular contractions followed each other rapidly, the corresponding diastoles were attended with a slight retraction of the auricles, most conspicuous at their sinuses. This phenomenon proceeded from the increased suction of blood by the ventricles.

6. *Are the auricles ever empty, or are they constantly full?*

A. Constantly full, their motions ranging between fulness and distention.

The following dictations formed a corollary.

" *The first and second sounds were heard, and the corresponding motions (the systolic and diastolic) were felt, while the auricles were not contracting.*"

Remark.—Had this observation been made in my first experiments, it would have superseded the necessity for much reasoning, as it conclusively fixes the sounds, the impulse, and the *back-stroke* or diastolic shock, on the ventricles.

" *When the heart was gorged, towards the conclusion of the experiments, the first sound only was heard.*"

Remark.—At the same time the action was very feeble. This, as in the experiment on the rabbit, displays the cause of the diminution of sound and impulse in suffocative dyspnœa, and on the supervention of death.

Sir B. Brodie, finding himself unable to attend these experiments on the 10th, favoured me with the following communication on the evening of the 9th. If doubt remains on the mind of any respecting the points in my experiments to which his observations refer, they cannot fail to have the weight which attaches to anything that proceeds from the pen of this distinguished physiologist.

My dear Sir,

. . . . With respect to some of your propositions, I think that I can already solve them in a way satisfactory at least to myself. 1. When I was making experiments on the circulation formerly, it appeared to me that the pulse and the systole of the ventricle exactly coincided. 2. It appeared to me that, when the action of the heart was vigorous, the ventricles emptied themselves at each contraction; but that they did not do so, when the action of the heart was feeble. 3. I never found the auricles completely empty themselves, nor did I, in dogs, rabbits, &c. ever observe in them any regular systole* corresponding to, and alternating with, that of the ventricles. I often used to observe several slight contractions of the auricle, especially of the appendix of the auricle, for one of the ventricle. In frogs, however, I have a strong recollection that the actions did alternate and correspond, but, not being able to find my notes, I cannot speak positively.

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* Sir B. Brodie's observations were here imperfect.

If I were to institute such a series of experiments, I would first stupify the animal by inoculating him with the woorara, or some poison of the same kind. You will observe that when an animal is stupified with the woorara, there is no struggling, and you may maintain the heart's action, by inflating the lungs, for an indefinite period. I have some woorara, and can, I doubt not, furnish you with enough for the experiment.

I am, dear Sir, yours very truly,
B. C. Brodie.

The woorara arrived too late for the experiments on the asses. I have already described how well it subsequently answered on a rabbit. Prussic acid was tried on one ass, but the animal recovered from four or five successive drachms given by the mouth, the poison being bad.

SECTION II.

EXPERIMENTAL, PHYSIOLOGICAL, AND PATHOLOGICAL RESEARCHES ON THE SOUNDS OF THE HEART.

Though the experiments detailed in the preceding section fixed the first sound of the heart on the ventricular systole, and the second, on the diastole, they did not go far enough to demonstrate the *immediate* cause of the sounds. In the first edition of this work, and previously in the Med. Gaz. of July 1830, I rejected the view that the second sound was occasioned by the closure of the semilunar valves, for the reasons above explained at page 13, which I subsequently found to be erroneous. I also rejected, as the cause of the first sound, the muscular "sound of rotation" (*bruit rotatoire*), resembling the rumbling of distant wheels, first described by Wollaston and Erman, and adopted by Laennec, as attending the contraction and braced state of muscles, and which any one may readily perceive by applying his palm to his ear while he alternately braces and relaxes his arm.

(For all the particulars, see Traité de l'Auscult. par Laennec, ii. p. 430, second edition). I rejected this (and I have had no reason to alter my opinion up to the present time), because, after torturing, and seeing others torture, muscles in every conceivable way, I never could succeed in producing, or thinking that others produced, a sound at all resembling those of the heart, either first or second, in shortness and clearness; for it must not be forgotten that the first sound, as well as the second, is short and clear in naturally thin hearts, and in dilatation of the organ. Nor has M. Bouillaud been more successful. After a great number of experiments, he says, "There is not the least resemblance between the rotatory sound and the sounds of the heart." Having thus excluded closure of the valves and rotatory rumbling as the causes of the sounds, I too hastily adopted an inferential explanation—that they were occasioned by the motions of the blood. This explanation, never quite satisfactory to myself, because not admitting of direct proof, was soon doubted by the profession. The valvular theory was revived by Dr. Billing in May 1832; subsequently by M. Rouanet, who frankly and handsomely avows that he derived the idea from Dr. Carswell in 1831; simultaneously by Mr. Bryan; afterwards by Mr. Carlile, and finally by M. Bouillaud, who adopted, with slight additions, the theory of Rouanet. To Dr. C. Williams, who had already embraced the theory which referred the sounds to "bruit musculaire," it still appeared that the most simple and satisfactory way of accounting for the first or systolic sound of the heart, was, to refer it to this class of sounds (Rational Exposit. Appendix to 2nd edit., p. 199, Sept. 1833); while he was "inclined to place the seat of the second sound in the parietes of the ventricles rendered momentarily tense by the sudden influx of blood." (Ibid. p. 201.)

Feeling it my duty immediately to correct my explanations, if erroneous, I commenced a new series of hospital researches on the living and dead subject, in 1832, without, however, being acquainted with the particulars of the valvular theories above alluded to, most of which, indeed, had not yet appeared. I cannot refrain from stating that the most able of the papers advocating this view and refuting my own, was published by Mr. Bryan, a sensible and vigorous writer, in the Lancet, August 6,

1833. He employs the bulk of the arguments, and especially a case of my own (Anderson), which had already led me to discard my old theory of the sounds. I was not aware of the existence of this paper till 1838.

By examining a vast number of patients in the St. Marylebone Infirmary, I speedily satisfied myself that the first sound was loudest over (or, as I subsequently found, below) the middle of the ventricles; and the second, over the sigmoid valves, and thence for a few inches upwards; also, that when a healthy subject was faint, the first sound lost its prolongation, and became short and smart like the second; whence I inferred that, in its natural state, it might have a compound cause, viz. the closure of the valves, and the motion of the blood, or the *bruit musculaire* or *rotatoire.*

The presumptions, thus offered, that the valves were concerned in the production of the sounds, required corroboration by experimental and pathological evidence. Not having succeeded in satisfactorily imitating the second sound by injecting fluids retrograde into the aorta, I tried the expansion of membranes under water, and found that three inches of fine tape, two lines broad, held to the end of a stethoscope, and gently jerked under water, imitated the second sound, both the sounds in dilatation, and the double sound of the fœtal heart, to perfection. Hence it was more than probable that the sudden expansion of membranes so small as the sigmoid valves was *sufficient* to produce such a sound as the second.*

It was not easy to meet with satisfactory pathological cases on this subject; as, to be conclusive, great disease of the valves on both sides of the heart simultaneously, seemed to be required. The case of Anderson was one of this kind; the mitral aperture being about a quarter of an inch, and the tricuspid half an inch in diameter; yet the second sound, though weak, was perfect and without a murmur. Now, had this sound been occasioned merely by the influx of the blood, or any other cause than the sigmoid valves, surely it would have been attended with a murmur.†

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* M. Rouanet's experiments on membranes, &c. correspond with my own. I tried every variety of membrane in breadth, extent, and thickness; also silk handkerchiefs, cloths, &c.

† In November, 1837, I saw a case the precise converse of this: namely, both sets,

R. S., Esq. whom I saw in consultation with Dr. Armstrong, had a prolonged bellows murmur over the sigmoid valves instead of the second sound. On examination, the orifice of the ossified and dilated aorta was found so much enlarged that the valves did not close it; hence resulted a murmur from regurgitation, which extinguished the second sound. But why, it will be said, was the sound not produced by the pulmonic valves? True; therefore I did not consider the case conclusive. I had elaborate notes of three other similar cases; but as the patients were living, the evidence was still less conclusive. I had, however, notes of the case of Thomas Wood, in the St. Mary-le-bone Infirmary, Oct. 21, 1834, made by myself and by Mr. Hutchinson, resident surgeon to the institution, separately, attesting that a murmur from regurgitation through the mitral valve *completely* drowned the first sound in the vicinity of the valve. Whence it might be inferred that a murmur in one set of sigmoid valves might possibly drown the natural sound of the other set. I have subsequently ascertained that, over the valves, this may actually be the case. But the natural sound of one set of sigmoid valves can always be heard by listening two or three inches up the vessel to which that set belongs, as its sound is thus withdrawn from the murmur produced in the other set.

On the whole, therefore, the presumptions were exceedingly strong in favour of the second sound being produced by the sigmoid valves.

The evidence on the first sound, more fully explained in the sequel, led me to establish the following presumptions; viz. that the first sound was *compound*, consisting, 1. Of the valvular flap; 2. Of an augmentation of this, either from *bruit musculaire*, or the motion of the fluid, or both;* 3. Of the prolongation of the sound by *bruit musculaire*, or the motion of the blood.

These presumptions required to be proved. No experiments had hitherto been devised which afforded direct demonstrative proof. M. Bouillaud, one of the last writers on the valvular

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of semilunar valves were disabled, and the second sound was almost completely extinct. These two cases afford as complete pathological evidence on the point as could well be desired.

* It will presently be seen that another cause, the sound of muscular extension, was the principal source of this augment.

theory, and who published in 1835, says, "I will begin by avowing that the proofs, direct or experimental, of the theory which we discuss, are almost completely wanting, and probably ever will be wanting." (*Traité*, I. p. 133-4). After much reflection, a mode of experimenting on the ass occurred to me in the autumn of 1834, which, if practicable, would inevitably, I thought, prove conclusive; namely, after denuding the heart in the manner described at page 14, to work out the following *Propositions*:—

1. *Is the second sound loudest over the sigmoids, and is it so* near *as to seem produced immediately under the stethoscope?*

2. *Is the first sound loudest over the two auricular valves respectively; and is it so* near *as to seem produced immediately under the stethoscope?*

3. *Place the origins of the aorta and pulmonary artery between the finger and thumb; apply the stethoscope on the heart near the sigmoids; instantly after the ventricular systole, close the arteries, so as to prevent the reflux of the blood and consequent expansion of the valves, and see whether this annihilates the second sound.*

4. *Relax the fingers during the interval of repose, and see whether this reproduces the second sound at its wrong interval.**

5. *Push the knuckle, or the auricle, into each auriculo-ventricular orifice, so as to prevent the expansion of the auricular valves, and see whether this annihilates the first sound.*

6. *Introduce a bent needle into the aorta, and hold open one or more of the semilunar valves, so as to permit free regurgitation. Notice whether this occasions a murmur with the second sound. The pressure of the aortic system being thus thrown on the ventricle, will it close the mitral valves? See whether this annihilates the first sound on that side.*

To pave the way for the performance of these experiments on the ass, I first made trial on a rabbit, poisoned with woorara, assisted privately and confidentially by Mr. H. James Johnson, lecturer on anatomy at Kinnerton-street, and one of the proprietors of that theatre. Though the heart acted vigorously for an hour, and we could *perfectly hear both sounds* by applying the small end of a thin stethoscope, the organ was too diminutive, and its movements too quick, to admit of our appreciating modifications of the sounds.

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* This could never be accomplished.

I then proceeded to a trial on the ass, at Mr. Field's, veterinary surgeon, Nov. 3, 1834. For the purpose of obviating any possible question respecting my title to the experiments as the inventor, I took the precaution on this, as on all former occasions, of putting a *written copy of the above Propositions into the hands of the friends invited,*—Dr. Davies, of Broad-street, Dr. C. J. B. Williams, Mr. H. James Johnson, and Mr. Field. My woorara being exhausted, I employed the hammer; but the instrument being too round-headed and the operator inexpert, the experiment failed, the action of the heart being nearly suspended by the time that the organ was exposed. The valves, indeed, were hooked back and the sounds heard, but with unsatisfactory results. The heart was also cut open, and its contractile movements were observed and listened to: finally, we carefully examined the situations of the valves, and *practised the hooking of them up*, with a view to a renewal of the experiments. I made arrangements to renew them immediately, and also requested, and was favoured with, a fresh supply of woorara by Sir B. Brodie. From unforeseen causes, over which I had no control, the experiments were delayed till the ensuing February. The woorara was then employed, and the heart, when denuded, beat with vigour and regularity about 60 or 70 per minute, and continued so to beat for an hour—affòrding ample leisure for making the following *Observations*, which answer to the above *Propositions*. The observations, with two or three exceptions only, were dictated and written by myself, under correction of the party, during the progress of the experiment. I publish them verbatim from the original manuscript, a rule to which I have always adhered. I therefore disclaim another version with verbal alterations, giving, in my opinion, a slight bias in favour of the exclusive muscular theory, which I believe to be erroneous.*

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* These experiments have been appropriated by a certain gentleman. At their second performance, on November 3, 1834, I promised him, from friendly motives, the *use* of them for a forthcoming edition of his book; and immediately before their final performance in February 1835, I permitted him, as he expressed a wish, to become my conjoint associate, and subsequently lent him my own notes of the experiments, written almost entirely with my own hand. He detained the notes, claimed

the experiments, and interdicted me from "*pirating*" them. An arbitration by Sir B. Brodie, and a final agreement through Dr. Macleod, decided that the experiments were conjoint, and that each should publish them as such. I did so (Appendix to second edition, page v): he appropriated them. As he has, by this measure, rejected the participation which I gave him, I resume my exclusive right as the sole inventor. His bold pretensions to the invention (to which he did not contribute directly or indirectly) are annihilated by a comparison of my *Propositions* with the results and an examination of the date (Nov. 3, 1834) when those Propositions were placed, in writing, in his hands; not to mention the previous confidential performance of the experiments with Mr. Henry James Johnson.

I am reluctantly compelled to make these remarks in consequence of advantage having been taken of a mistake in Sir B. Brodie's arbitration to show it to my disadvantage. Sir B. Brodie mistook the question, and arbitrated as if I had interdicted the opposite party, and not he me. Notwithstanding, the result was entirely in my favour; for he decided (as I contended) that the experiments were *conjoint*, not even questioning *my* right to publish them: and he further bound the opposite party "carefully to explain what share he (Dr. Hope) had in projecting and planning the experiments in the first instance."

The following is from Dr. Thomas Davies, who assisted at the experiment, November 3, 1834.

My dear Sir,

I can truly say that, although I had read of the movements of the valves being the cause of the second sound, yet yours were the first experiments I ever saw or heard of, for the purpose of attempting to prove it.

I remain, yours very truly,

Thomas Davies.

New Broad-street, July 21st, 1835.

8, Suffolk Place, January 14, 1839.

My dear Hope,

I can have no hesitation in stating, that I first assisted you in making some experiments on rabbits, to determine the causes of the sounds of the heart, in the spring of 1830. These experiments were performed in the rooms of the house-surgeons of St. George's Hospital.

The second occasion on which you did me the honour to ask my assistance, was in the autumn of 1834, when we operated privately on a rabbit at your own house. On this occasion you produced (to the best of my remembrance) the paper of "Propositions" to be verified or disproved, which you have enclosed to me, and I never doubted that they were your own.

It was November 3, of the same year, that I was again present at a repetition of the former experiments, at Mr. Field's, when the paper, which I believe to have seen previously, was reproduced.

Subsequently to this, my colleagues and myself had the pleasure of placing the dissecting-room in Kinnerton-street at your disposal, for a continuation of the experiments on animals, at the first of which (for there were more than one) I was present, but not at the remainder.

Yours very truly,

Henry James Johnson.

Notwithstanding what precedes, the individual alluded to writes as follows, in the last edition of his Pathology and Diagnosis of Diseases of the Chest, p. 169: "I concluded the Appendix (to the previous edition in 1832) by recommending Dr. Hope to investigate experimentally these points (the sounds of the heart), which were there shown to be doubtful; but his engagements and other circumstances having *prevented him from doing so,* I lately undertook the task; I made a point of ensuring Dr. Hope's presence and testimony at the experiments, and I conferred with him and several other gentlemen as to the best mode of performing them. *I was present at an experiment attempted by Dr. Hope, in November last, at Mr. Field's; and he then planned modes of suspending the action of the valves, similar to some of those afterwards adopted in my experiments.*" The last sentence was added, I presume, to meet the order in Sir B. Brodie's arbitration. How far it does so, and whether it is not rather an indirect appropriation of the *invention* of the experiments, the reader can judge.

SERIES I.*

Obs. 1. The first sound was perfectly loud and distinct; and it was louder on the body of the ventricles than over the semilunar valves.

2. The second sound was more audible over the semilunar valves than at the other parts of the heart, being sometimes dis-

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* Present—Drs. Arnott and Williams, and Messrs. Babington, Smyth, H. James Johnson, Peregrine, Good. Messrs. Charles Johnson and Tatum were temporarily present.

These experiments were repeated, and republished a year and a half afterwards, by the London Committee of the British Association. They verified the whole, but without eliciting anything new beyond a few illustrations and corroborations, which I shall append in the form of notes to this and the following series. This sameness is, perhaps, to be ascribed to the circumstance, that one of the committee, who, as Dr. Todd informed me, directed the others in everything, had been present at the whole of my experiments. It was probably from the same cause that the committee adopted the *pure muscular theory* of the first sound, previously advocated by the same individual, and from which I dissent as being too exclusive.

"The subjects of their observations, say the committee, were, in most instances, young asses, from three to six months old, apparently in good health; and the mode of operation was, in a few instances, poisoning with woorara; in others stunning by a blow on the head; but in the majority, the animal was pithed."

The successful experiments were fourteen in number, but the committee say that they were "much less fortunate than several preceding experimentalists, having, in no one subject, been able to continue their observations for a longer period than forty-five minutes." The report is ably drawn up by Dr. Clendinning.

tinct at the mouths of the arteries when inaudible on the body of the ventricles.

3. Pressure on the arterial orifices by the fingers or the stethoscope invariably stopped the second sound. Slight pressure caused a whizzing or bellows murmur with the first sound.*

4. The first sound was diminished, but not wholly suppressed, by pressing upon the ventricles with the end of the stethoscope (so as to curb or restrict their full contractile tension).

5. At each systole the sudden tension of the ventricles was such as to produce an *abrupt shock* to the finger placed on any part of them, with which shock the first sound exactly coincided. (This observation was pointed out by the writer).†

6. The first sound was diminished, but not suspended, by thrusting the ends of the fingers into the auriculo-ventricular orifices; the ventricles contracting less, and irregularly (from the impeded influx of blood).‡

7. An incision being made into the left auricle, and the scalpel being passed into the ventricle, so as partially to destroy the mitral valve, and the blood being allowed freely to escape, the first sound continued to be heard with each contraction of the ventricle. See 9 *a*.

8. The sound continued, though the right auricle was completely cut open.

9. And, finally, though the finger was introduced into the left

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* The committee observed that heavier pressure caused "a loud bellows- or rasp-sound" (Exp. 7). The same occurred in my *Obs.* 14.

† The committee say "at each systole, while the heart acted vigorously, the ventricles felt to the finger *as hard as cartilage*." Again, "The tension and hardness of the ventricles during their systole were very remarkable" (Exp. 14).

‡ The committee here say "A first sound was heard, prolonged by a whizzing murmur" (Exp. 9 and 12). Again, "The inversion of the auricles was accompanied with a sensation of *thrilling* in the finger of the operator, synchronous with the impulse" (Exp. 10 and 12). The murmur and thrill, I had noticed a year previously (See Obs. 26, in the third Series). Also, "On inverting the auricles again, the chordæ tendineæ of the mitral valve alone, were felt to become tense in systole, and lax in diastole" (Committee Exp. 12). This observation militates against the experimenters' own theory: viz. that the first sound is purely muscular and independent of extension of the valves and their chordæ. Again, "The finger being introduced into the left ventricle by inversion of the auricle, was felt to be gently embraced and pushed, as if by a membrane distended with blood. On the right side, nothing similar was unequivocally observed" (Exp. 14). This observation also, by showing the tense state of the membrane of the valve, opposes the theory of the experimenters.

ventricle, and was made by pressure to prevent the influx of blood into the right;

a. Its character, however, was not so clear and smart as when the ventricles contracted on their blood;*

b. Thirty or more contractions, the majority very vigorous, took place after the incision had been made.†

SERIES II.‡

Obs. 10. Before the pericardium was opened, *both* sounds were very distinctly heard.

11. Both were also distinctly heard through the lung interposed between the heart and the end of the stethoscope.||

12. About two or three inches up the aorta from its origin, the second sound was heard (but not the first), alternating with the impulse as felt on the ventricles. (This observation was suggested by myself (not by Mr. Keate), in consequence of my having previously noticed the same in the living subject, as stated at p. 27).

13. The second sound was decidedly more distinct over the origins of the aorta and pulmonary artery than on the body of the ventricles; and, in that situation, it was louder than the first sound at the same point. It had exactly its natural short, clear, flapping character.

14. The aorta and pulmonary artery being compressed between the fingers, the first sound was accompanied with a loud murmur, and the second was stopped.

15. A common dissecting hook was passed into the pulmonary artery, so as to prevent the closure of the semilunar valves: the second sound was impaired, and a hissing murmur accompanied

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* The committee say "The first sound was still distinctly heard by all, but *weak* (Exp. 7).

† I had made incisions in *all* my previous experiments, both in 1830, 1831 and 1834. The idea, therefore, though not specified in the *propositions*, cannot be claimed by one who had attended those experiments.

‡ Present Drs. Williams and Macleod, and Messrs. Keate, Partridge, Malton, Goode, Seagrim, and others who looked in only temporarily—including Mr. Henry Johnson. Mr. Tatum was absent. The heart acted vigorously for an hour.

|| I made this observation to refute the contrary opinion held by M. Majendie.

it. A hook was passed into the aorta, so as to act in the same way on the aortic valves: the second sound entirely ceased, and was replaced by a prolonged hissing. (Heard by several.)

16. When the hooks were withdrawn, the second sound returned and the hissing ceased.

17. Experiment 15 was repeated, and whilst Dr. Hope listened, the hook was first withdrawn by Dr. Williams from the aorta. Dr. Hope immediately said, "I hear the second sound."

18. Dr. Williams then removed that from the pulmonary artery; Dr. Hope said, "the second sound is stronger, and the murmur has ceased." (Several listened to 16, 17, and 18.)

19. The arteries were cut open: the heart continuing to contract (about eight or ten times), the first sound only was obscurely audible.*

SERIES III.

On August 7th, 1835, six months after the two preceding series, I performed the following, at the Kinnerton-street Theatre, assisted by Dr. Latham, physician to St. Bartholomew's, Dr. Watson and Mr. Mayo, physician and surgeon to the Middlesex Hospital, and Mr. Thorpe, a student of St. George's Hospital. My principal object was, to ascertain with more precision, to what extent the auricular valves were subservient to the production of the first sound, and I had projected a new experiment (see below, Obs. 26) with this design.

A large ass, aged 8 or 9, with a pulse at 50, was employed. Having formerly found six or eight grains of woorara barely sufficient to kill an animal, and generally in not less than half an hour, and having only two grains of the poison remaining, I adopted the following process, which was singularly successful, the heart having acted no less than two hours after the death of the animal, which is an hour longer than in any other experiment with which I am acquainted, except those of the Dublin Committee of the British Association, performed about the same time, August 1835. "The period," they say, "varied in different subjects from one to two hours."

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* The committee say "The pulmonary artery being cut across, the first sound was still loud: and the aorta being then cut across likewise, the same result was obtained —viz. a first, without a second sound. The heart was then severed from its several attachments, and the systolic sound was still heard distinctly. The heart was then grasped strongly under blood: it continued to contract vigorously, and the first sound was heard (but not loud) with the flexible tube as well as the common stethoscope. The heart was then taken out and held in the hand of one of the committee; when the first sound was distinct, but feeble. On opening the right ventricle, the columnæ carneæ were distinctly seen contracting simultaneously with the ventricle" (Exp. 14).

The jugular vein having been denuded by Mr. Mayo at the ordinary bleeding point, which is the most superficial part of the vein, and the finger having been passed round it, a small incision was made in the vessel, just sufficient to admit an ounce-syringe charged with a solution of the two grains of woorara in an ounce of water. This being injected, the aperture was pinched up with the forceps and secured by ligature. Respiration *instantly* began to fail, in less than a minute it had nearly ceased, and in a minute, wholly. The trachea was then opened, a bellows-pipe introduced, and artificial respiration established. The chest was next opened by sawing through the ribs (which were ossified) close to the sternum, and making two incisions along the intercostal spaces towards the spine, so as to include three or four ribs, which were then broken backwards. Large vessels were secured in the course of the operation to prevent hemorrhage, because, by taking off the tension of the vascular system, it diminishes the loudness of the sounds.

The heart, when denuded, was beating steadily and with surprising power, about sixty per minute, and it continued to beat between forty and seventy per minute for two hours, the frequency increasing above sixty, whenever, from diminishing the inflation or from compressing the lungs with the hand, the supply of blood to the left side of the heart was insufficient, which was indicated by the flabbiness and diminished size of the left ventricle.

The results (which I have numbered consecutively with the two preceding series) were as follows:—

Obs. 20. *The first sound is audible through interposed lung.*

21. *The second sound is loudest over the sigmoid valves, and for two or three inches along the aorta and pulmonary artery.*

22. *The first sound is loudest over the parts of the ventricles nearest to the auricular valves.*

Having in my written *Propositions* for the present series requested the experimenters to "make observations on the shock of the ventricles at their *maximum* tension," the following was the answer:—

23. *The impulse from lateral expansion was greatest at the* margins of the auricular orifices, *there throwing the finger out with a violent jerk. The lateral expansion of the base (i. e. upper part) of the ventricles attended the retraction of the apex.*

24. *On lightly placing the finger and thumb on each side of the pulmonary artery, opposite to the valves, a shock corresponding with the closure of the valves was distinctly and repeatedly felt by Dr. Latham, Mr. Mayo, and myself (Dr. Watson having retired); and I felt the same on applying my finger to the aortic valves.*

The same observation was made, a year later, by the London Committee of the British Association, but they had the priority of publication in their Report, August 1836. Their words are, "Immediately after the systole, a flapping or jerking sensation was sensible to the finger applied to the arteries at their roots" (Exp. 10). Again, "on touching the arteries in the vicinity of the valves, a sensation of flapping (or jerking) was observed by all, commencing immediately after the systole, and accompanying the second sound" (Exp. 13).

This observation *proves* nothing, but it corroborates others by conveying to the mind a strong *feeling* or impression of the force and smartness with which the semilunar valves close, and of the sufficiency of such membranes, so closed, to produce such a sound as the second.

25. *A hook was passed into the pulmonary artery and the valves held open. This created a sighing-murmur instead of the second sound, previously very distinct. The hook being withdrawn, the murmur ceased and the second sound returned as distinct as before. This was repeated three times with the same result.*

On the last occasion the hook got entangled, and on withdrawing it, the sighing-murmur, with diminution of the second sound, *continued permanent; which I predicted to indicate an injury of the valve, as was subsequently found to be the case;*

This valve being injured, and about an hour having elapsed, I did not venture to hook up the aortic valves, lest I should be prevented from making the next observation (No. 26), which was the main object of the experiment, and from which I hoped for important results. On formerly trying my *Proposition* No. 5, viz. "push the knuckle or the auricle into each auriculo-ventricular orifice, so as to prevent the expansion of the valves, and see whether this annihilates the first sound," I had found, in Series I. and II., that pushing so large a body as the auricle into the orifice, prevented the influx of blood, and thus disturbed the regularity and completeness of the heart's contractions: I therefore devised the following mode of accomplishing the same object.

26. *I passed a needle of flexible wire through the insertion of the left auricle, and out at the opposite side, and then bent the needle into the ventricle, so as to prevent the sudden expansion and closure of the mitral valve.*

This greatly diminished the first sound and created a very loud murmur from regurgitation, which I also felt to be attended with a strong thrill (fremissement cataire) at the margin of the auricular orifice.

Hence the loudness of the first sound is *connected* with the closure of the valves, but this experiment does not prove that the valves are the *sole* cause of the sound, because the regurgitation would diminish the tension of the muscular walls and, consequently, the sound produced by it. We occupied an hour in making this single observation, trying it in every way and with the utmost care; when the action of the heart suddenly and spontaneously stopped, and put an end to the experiment.

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Autopsy.

One of the pulmonic valves presented an oval aperture from laceration by the hook, capable of admitting a large quill, thus realizing my anticipation that this was the cause of the permanent murmur from regurgitation.

An interesting appearance was noticed along the margins of the aperture and at several points of the pulmonary artery and interior of the ventricle, where the lining membrane had been lacerated by the point of the hook: namely, the parts were overspread with a number of pink, semitransparent, fibrinous coagula, the size of pins heads, and easily removeable with the handle of the scalpel. Their characters, in short, were closely analogous to those of ordinary valvular vegetations. It may be a question whether they proceeded from inflammatory exudation of lymph, or from mere entanglement of blood by the broken surface. The former view is perhaps the more probable.

CONCLUSIONS FROM THE WHOLE OF THE EXPERIMENTS ON THE SOUNDS.

CONCLUSIONS ON THE FIRST SOUND.

I shall not notice those theories which ascribe the first sound,

1st. To the *collision of the particles of the blood against each other and against the walls, in the interior of the heart* (formerly broached by myself, but discarded in 1832).

2d. To the *collision of the opposite interior surfaces of the ventricles at the conclusion of their systole*—both these theories having been sufficiently disproved.

The theory of M. Majendie is, that *the first sound is occasioned by the collision of the heart against the ribs during its systole, and the second, by its collision against the sternum during the diastole.* This theory is completely refuted, 1st, by my two original series of experiments on the ass in 1830 (see p. 14), proving that the sounds were perfect when the sternum and ribs were removed: 2d, by my foregoing experiments on the sounds, Obs. 11, in which "both sounds were distinctly heard through the lung interposed between the heart and the end of the stethoscope"—an observation which I made specifically to refute M. Majendie, and which was subsequently verified by the London Committee of the British Association: 3d, it is refuted by hydro-pericardium; as the sounds are perfect though the interposed fluid prevents the heart from impinging against the walls of the chest. This theory, I should have dismissed more summarily, but that I perceive it has been partially admitted by the London Committee of the British Association, and by one of its members in a separate publication. "An extrinsic or subsidiary sound," say the committee, "which, in a variety of circumstances, contributes largely to the first sound, arises from the impulse of the heart against the parietes chiefly of the thorax;" and Dr. W——, in his separate publication, says, "I am disposed to admit that, in violent action of the organ, its more sudden and abrupt strokes against the chest *do* cause a sound, which constitutes the loud termination of the first sound in these cases, and which seems nearer the ear, and more like a knock, than what is heard in the ordinary action of the heart. In common pulsations, the apex of the organ is drawn upwards and forwards at each systole, and sliding obliquely on the smooth pericardium, does not impel against the ribs with sufficient abruptness to cause sound. But in quicker and more violent pulses, the abruptness of the motion, and the force of the *blow* against the side of the chest, are such as can scarcely fail to produce sound" (On Diseases of the Chest, p. 178).

These conclusions are precipitate. They proceed on an assumption which is doubtful at least, if not absolutely erroneous: namely, that the heart "*impinges* upon" (committee), or gives a "*blow* against" (Dr. W——) the chest. Now, as has indeed been well remarked by Mr. Bryan (Lancet, vol. xxix. p. 501), the heart is held in contact with the walls of the chest by a force of fifteen pounds to each square inch: if held in contact, it cannot *impinge* or strike a *blow*: it can merely heave the chest, and such heaving, however sudden or powerful, cannot produce sound. The committee rest on the fact that "leaning" to the left or forwards gave additional force to the impulse and loudness to the sound; while inclination of the body, such as to cause the heart to gravitate away from the ribs, diminished at once the "sound and impulse." But in both these positions, the heart is still in contact with the walls of the chest, as is proved to demonstration by the continuance of *dulness on percussion*,—impaired indeed by leaning backwards, but increased by leaning forwards. If, therefore, the heart is more decidedly and positively in constant contact with the chest on leaning forward, that is precisely the time when the increase of sound observed by the committee ought *not* to be referable to the organs "*impinging*" or "*striking* a *blow*" against the chest. This argument alone is conclusive against the doctrine in question; but as the committee attaches much weight to the following experiment, it may be desirable to point out its inconclusiveness.

"When a small opening," say they, "was made in the cartilages opposite to the heart, the heart during systole was felt, both outside and inside the chest, to *press* with force against the sternum and cartilages." But here, if air was *not* admitted, and the heart was kept in contact with the walls by atmospheric pressure, the observation proves nothing to the point; as it proves, not a *blow*, but merely "*pressure* with force against the sternum"—which pressure is insufficient to produce sound. But if air *was* admitted, the observation still proves nothing; as the air, by causing collapse of the lung, created any interval that existed between the heart and the walls of the chest.

But though it is thus proved that the first sound does not receive an augmentation from the heart impinging against the walls of the chest (a conclusion to which I may here state that the Dublin Committee of the British Association also came, in 1835); yet it *does* occasionally receive an augmentation from another cause, the nature and circumstances of which have been overlooked, not only by the committee, but, I believe, by every other writer up to the present time; and this same cause is the source of the *metallic cliquetis* or *tinnitus* of Laennec, the true explanation of which, for the same reason, has never been given. The cause alluded to is simply this: the heart in gliding forwards and upwards during its systole, strikes with its apex against the *inferior margin* of the 5th rib, and thus creates an accidental sound, attended with cliquetis when the blow is smart. It may be prevented at pleasure by pressing the edge of the stethoscope or anything else into the intercostal space, by which that space is put, internally, on the same plane as the rib, over which the heart then glides without catching. I have never found the sound to occur in any but the meagre; because, in the well-conditioned, the intercostal spaces are full and resistent, and, consequently, the edge of the rib is not exposed. It is not necessary here to dwell on this phenomenon,* but I may remark that I have for many years noticed the first sound to be *double* in some patients.

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* See case of Carrington, Allan, &c.

The cause is, that the blow of the heart against the edge of the rib is a little later than the first sound. As the costal sound is *accidental*, it cannot be considered as constituting a part of the normal first sound of the heart.

The necessity for an augment from extrinsic sources experienced by the London Committee, perhaps originated in a difficulty under which the advocates of mere bruit musculaire as the cause of the first sound, found themselves: namely, that this sound, during palpitation, is in some instances of such extraordinary intensity, that it would do violence to all analogy to suppose it produced solely by bruit musculaire. The sounds of muscular and valvular extension, as will presently appear, adequately account for this intensity, and supersede the necessity of resorting to any extrinsic cause.

I now proceed to notice what the foregoing experiments, as well as pathological observations, lead me to regard as the exclusive causes of the first sound: namely,

1st. *The sound of muscular extension.*

2d. "*Bruit musculaire ou rotatoire*" — the dull, rumbling sound of muscular contraction.

3d. *The sound of valvular extension*, the most important of all.

I place the causes in the above order, not from their relative importance, but as the most convenient for discussion.

First Sound, how far caused by Muscular Extension.

By the term "sound of muscular extension," which I have used to avoid circumlocutions, I mean a loud, smart sound produced by the abstract act of sudden, jerking extension of the already braced muscular walls, at the moment when the auricular valves close; in the same way that, when the valve of a pair of bellows closes, its leather is put on the stretch, and, if not rigid, produces sound.* Further, by the sound of muscular extension I mean a phenomenon essentially different, in my opinion, from *bruit musculaire;* since the extension-sound may be produced even in a dead muscle and may attain a high degree of loudness and smartness, whereas bruit musculaire can only be produced in a living muscle and is never more than dull and subdued.

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* Dr. C. Williams has remarked on my sound of extension, that "*not aware of the physical cause of muscular sound*, he (Dr. Hope) has called its abrupt commencement a sound of *extension*, which term, applied to *contracting* muscle, is obviously contradictory and erroneous. The cause of sound is resisted motion; and the strongest and quickest motion, most abruptly and forcibly resisted, will give the loudest sound" (Med. Gaz. vol. xvi. p. 820). This is a mere verbal criticism. The *fact* is that resisted muscular contraction is muscular extension, and this is all that I contend for. Who will deny that muscular contraction is resisted when the auricular

The existence of the sound of extension appears to me to rest on strong grounds, and the London Committee of the British Association, after repeating my experiments, are of the same opinion; for they say, "the facts relating directly to muscular *tension* as a possible cause of the first sound, are few but striking and in their judgment decisive."

The grounds are as follow: In Obs. 5 of my experiments, it was found that *at each systole, the sudden tension of the ventricles was such as to produce an abrupt shock to the finger placed on any part of them, with which shock the first sound exactly coincided.* This phenomenon, pointed out by myself and which awakened in me the first idea of the sound of muscular extension in contradistinction to bruit musculaire, made a forcible impression on all present; and it was remarked that the sense of touch conveyed an identical idea with the sense of the hearing, for the sound was as smart, loud and clear as the shock was abrupt. Again, Obs. 23. *The impulse from lateral expansion was greatest at the margins of the auricular orifices, there throwing the finger out with a violent jerk.* Again, the committee say, "at each systole, while the heart acted vigorously, the ventricle felt to the finger *as hard as cartilage*" (Exp. 4); and, "the tension and hardness of the ventricles during their systole, were very remarkable." (Exp. 14). Further, the Dublin Committee, August 1835, say, "the ventricles, with a *rapid* motion, assumed a somewhat globular form in their middle part," and "during their continuance in this state, they were *hard* to the touch, and, if grasped by the hand at the *commencement* of the movement, they communicated a *shock* or *impulse*, and separated the fingers" (Exp. 1). Under these circumstances, in all the observations, the first sound was perfectly loud and distinct, as described in my Observations 1 and 10.

But when the circumstances were altered, namely, when the

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valves close? That not only tension, but *extension* is occasioned at the moment of this resistance, I shall presently show.

resistance of the valves was removed, and the ***sudden shock or jerk*** of muscular extension thus prevented, the first sound was dull and obscure, like the muscular sound which may be imitated by the hand. This was exemplified in Obs. 7, " when an incision was made into the left auricle, and a scalpel passed into the ventricle, so as partly to destroy the mitral valve, and allow the free escape of the blood;" in Obs. 8, when "the right auricle was completely cut open;" in Obs. 9, when "the finger was introduced into the left ventricle, and made by pressure to prevent the influx of blood into the right;" and in Obs. 19, when "the arteries were cut open." In all these cases, the first sound "was not so clear and smart as when the ventricles contracted on their blood;" it "was obscurely audible." All this is admitted, with his usual inconsistency, by Dr. C. W——, a commentator on my experiments and an advocate of the exclusive muscular theory. He says the sound presented "the duller and more obscure character of common muscular sounds" (Med. Gaz. vol. XVI. p. 820).The London Committee, repeating the same experiments, say " the first sound was still distinctly heard by all, but *weak*" (Exp. 7); yet they have overlooked this weakness in their conclusions; for they inconsistently say, " *The unvarying* and *uniform* character of the systolic sound, however diversified the circumstances in which the heart was placed, furnishes a strong argument in favour of its intrinsic nature."

Such are the grounds on which the sound of muscular extension appears to me to rest. But it may be objected that the sound was occasioned, not by muscular, but by valvular extension. This argument is fair, and in fact it is difficult to say precisely to what extent the valves do take part in the production of the sound. Yet I believe that they do not produce it entirely, because the first sound of the heart during palpitation is, in some instances, (and here I do not allude to the accidental costal sound and metallic cliquetis described above at p. 41) of such extraordinary intensity, that it would do violence to all analogy to suppose it produced by extension of the auricular valves alone; and further, the loud sound of palpitation is of a more blunt character than the valvular click, such as I shall presently describe it as produced by the auricular valves.

First Sound, how far caused by Bruit Musculaire.

This term applies, in my opinion, to the sound, such as we heard it whenever valvular and muscular extension were removed by destroying the auricular valves or evacuating the blood out of the ventricles: namely, a *dull, obscure* sound (Obs. 9. 19), like the muscular sound which may be imitated by the hand, or, to use Dr. W——s' expression, presenting the "duller and more subdued character of common muscular sounds." This *bruit musculaire* may possibly augment the intensity of the sound, and also impart to it a dull or blunt character. It likewise contributes the well-known prolongation, which so forcibly struck the Dublin Committee, as to lead them to conclude that "the cause of the first sound is one which begins and ends with the ventricular systole, and is in constant operation during the continuance of that systole" (Report, August 11, 1835). It will presently be shown, however, that the first sound is not always prolonged; but that it is sometimes a mere click, and that this is probably in consequence of the absence of bruit musculaire.

First Sound, how far caused by extension of the Auricular Valves.

Under the word "valves," I include the chordæ tendineæ,—fine chords, equally calculated to produce sound as the membranous expansions of the valves.

I have stated above that it is difficult to separate the valvular, from the muscular sound of extension; because, being synchronous, they are, as it were, incorporated together. The reality, however, of the sound of valvular extension, appears to me to rest on the strongest possible presumptive evidence. In Obs. 22, "the first sound was loudest over the parts of the ventricles nearest to the auricular valves." I do not wish, however, to attach too much weight to this observation. Again, we have seen that the ventricles, in their systole, attain a hardness like that of "cartilage:" add to this Obs. 23; "the impulse from lateral expansion was greatest at the *margins of the auricular orifices,* there throwing the finger out with a violent jerk." Now, the margins of the auricular orifices, into which the valves are inserted, could not be jerked out with a power that renders the ventricles as hard as cartilage, without suddenly putting the valves and chordæ tendineæ on the full stretch. No one, I think, who carefully examines the anatomy of the auricular valves, will doubt this. Nay, it seems to have been actually felt by the London Committee. "On inverting the auricles again, say they, the chordæ tendineæ of the mitral valve alone, were *felt* to become *tense in systole* and lax in diastole" (Exp. 12). Also, "the finger was felt to be gently embraced and pushed, as if by a membrane distended with blood." Sound, therefore, must necessarily be the result of this violent valvular extension; for it has been proved by the foregoing experiments that sound positively is produced by smaller membranes,—the semilunar valves, acted upon with certainly inferior force, namely, that of the aortic column of blood.

Again, when valvular extension was prevented by holding the mitral valve open with a bent wire, as in Obs. 26, "this greatly diminished the first sound;" and whenever the auricular valves were destroyed or the blood evacuated out of the ventricles, the sound became dull and obscure.

This experimental evidence is corroborated by pathological. There is a considerable class of cases—some of valvular disease, others of dilatation with attenuation, others again of mere softening,—in which the ventricles contract three, four, or more times for each pulse felt at the wrist. Now, these intermediate contractions, though so feeble as to produce little or no pulse, produce a sound, and that sound is as brief, smart and clear—as pure a *click*, as is produced even by the semilunar valves. I believe, therefore, that it is occasioned solely by the extension of the auricular valves and chordæ tendineæ, for the production of which extension the feeble ventricular contractions are sufficient, though insufficient to produce either the sound of muscular extension or bruit musculaire.

The same argument applies, though in a less degree, to hearts with naturally thin walls, in which the two sounds approximate in quality (Laennec); and to dilatation with attenuation, in which they become absolutely identical. In both these cases, the feebleness of the ventricular contraction is evinced by the deficiency or absence of impulse; yet the first sound is a well-marked click. I put it as a question whether the first sound of the right ventricle is not, for the same reasons, smarter than that of the left. It is bold to be too confident on this point, as Laennec pronounced the sound of the two sides to be "similar and equal;" yet reiterated observations incline me to believe that the question which I have propounded, will eventually be answered in the affirmative. Bouillaud thinks the two sounds clearer at the base of the sternum than between the cartilages of the 5th and 6th left ribs (Traité I. 106).

Further, the reality of the sound of valvular extension is corroborated by the inability of the advocates of the exclusive muscular theory to rid themselves of the valvular sound, without direct contradiction. Thus Dr. W——, in one of his deductions from my experiments, says:—"That the first sound is produced by the muscular contraction itself, may be considered as proved by Obs. 8 and 9, in which every other possible source of sound was excluded, and the first sound still accompanied the systolic action of the ventricles" (On Diseases of the Chest, p. 175). Also: "That the first sound is not dependent on closing of the auriculo-ventricular valves, is evident from five observations, in which the closure of these valves was partially or completely prevented, yet the first sound continued" (Med. Gaz. Sept. 9, 1835). These deductions are, 1st, not only inconclusive, but actually opposed to Dr. W——'s theory; because the sounds, though not annihilated, were always *modified*—rendered dull, by disabling the valves: 2d, they are directly contradicted in the next page by Dr. W—— himself: namely, "probably, in common pulsations, the ventricles do not attain the degree of tension which is sonorous, until the closing of the auricular valves; this closure, as the commencement of the resistance, brings at once to its acmé the muscular tension, which continues until the contents of the ventricles are sufficiently expelled. This accounts for the sudden or flapping commencement often perceptible in the first sound, and it suggests how the *due action of the auricular valves generally contributes to its clearness. Thea uricular valves, the chordæ tendineæ, the columnæ carneæ, and internal fibres of the ventricles, if they attain the same degree of tension as the exterior of the ventricles, may have an equal share in the production of the first sound*" (On Diseases of the Chest, p. 177). No advocate of the valvular theory contends for more.

The London Committee of the British Association, of which Dr. W—— was a member, conclude "that valvular action is not a cause of the first sound," because that sound continued, though the action of the valves was prevented. But this conclusion is illogical, because, as already stated, the first sound was always *modified* by interfering with the valves. Their argument No. 2, "that the action of the mitral valve as felt by the finger, was of too gradual and feeble a kind to be capable of producing sound," is contradicted by another observation of their own which they seem to have inadvertently overlooked; viz. that the chordæ tendineæ of the mitral valve alone, were felt to become *tense* in systole" (Exp. 12). The committee endeavour to fortify their opinion by referring to the heart of the domestic cock, in which there "is no tricuspid valve resembling that of men, but the valvular office is discharged by laminar extensions of the substance of the parietes of the ventricle, which meet in the middle, so as during the systole to cover the auriculo-ventricular orifice:"—yet in this animal M. Bouillaud heard both sounds of the heart. This argument is without weight; for if the valve consists of laminæ at all, those laminæ, whatever their configuration, would produce sound when violently and suddenly extended.

The committee further endeavour to defend their exclusion of valvular sound, by the following experiment, tending to show that muscular contraction alone is adequate to the production of the first sound (Exp. 2). "From the abdominal muscular contractions, sounds of a systolic character in all respects, and as loud as, or louder than, those of the heart, were with facility obtained. The sounds were produced by sudden expiratory efforts made with force and with the mouth closed, and were obtained with the flexible ear-tube from various parts of the parietes. At the time the sound was heard, the muscle under observation always felt to the finger tense and hard; *but the sound ceased at the moment that the fibres had attained their maximum of tightness and hardness*, and was not renewed except by a repetition of the contractile efforts after previous relaxation."

I repeated this experiment, and found that the sound was owing, not to the muscular contraction at all, but to a trifling adventitious circumstance rather amusingly overlooked by the committee.

Making use of a flexible stethoscope, with a thin ivory extremity, such as I had seen used by the reporter of the committee,—in short, a common flexible ear-trumpet, I found that the "systolic" sound was produced to admiration on *some* applications of the ivory cup, but not at all on *others.* On carefully scrutinizing the cause of this remarkable difference, I found that the sound was produced by nothing more than the skin being drawn out of universal contact with the edge of the cup by the sudden retractile tension of the abdominal muscles; whence air was admitted and the sound generated on the same principle as when a cupping-glass is removed: for, in fact, the softness of the abdominal integuments, by causing them to rise into the cup, creates a slight degree of vacuum. Accordingly, I found that when the cup was applied and then suddenly lifted off, *without any motion whatever of the muscles of the abdomen,* the "systolic" sound was still produced as perfectly as ever. This experiment, therefore, which the London Committee have placed, as if triumphantly, in the van of their report, is a mere mistake.

The Dublin Committee of the British Association for 1835, conclude (No. 4) "That the cause of the first sound is one which begins and ends with the ventricular systole, and is in constant operation during the continuance of that systole." This may be controverted by a denial that the fact is universally true. The first sound, in hearts naturally thin or morbidly dilated, may, as already explained, be as brief as the second. This fact supersedes their next conclusion, No. 5, viz. "That it does not depend on the closing of the auriculo-ventricular valves, at the commencement of the systole, because such movement of the valves takes place only at the commencement of the systole, and is of much shorter duration than the systole."

I have now offered experimental evidence to prove that the closure of the auricular valves causes sound: I have shown that the advocates of the purely muscular theory of the first sound, admit the valvular sound *indirectly* though they deny it *directly:* finally, I have demonstrated that muscular sound alone is totally insufficient to account for the first sound when it possesses a short, clicking character like the second.

My conclusions respecting the causes of the first sound may therefore be summed up in the following terms:—

The first sound is compound, viz. consisting, 1st, of valvular sound; 2nd, of the sound of extension—a loud smart sound, produced by the abstract act of sudden, jerking extension of the braced muscular walls; 3rd, a prolongation, and possibly an augmentation, by *bruit musculaire.*

Now these conclusions are identical with those which I published in March 1835 (see the Appendix). We next proceed to the second sound.

CONCLUSIONS ON THE SECOND SOUND.

As my experimental evidence amounts to demonstration, that this sound is produced by the closure of the sigmoid valves exclusively, I may here be brief.

The evidence is, 1st, that compression of the arterial orifices with the fingers, so as to prevent the reflux of the blood on the semilunar valves, invariably annihilated the second sound (Obs. 3 and 14, and the same repeated by the committees).

2nd. Hooking up a semilunar valve in one artery, invariably diminished the second sound; and doing the same simultaneously in both arteries, invariably annihilated it, a murmur from regurgitation being produced in its stead (Obs. 15, 16, 17, and 18, and the same repeated by the committees).

3rd. The second sound was loudest on the sigmoid valves and thence for two or three inches up the aorta. The conclusions may be summed up in the following terms:—

The second sound is produced by the sudden expansion of the semilunar valves, resulting from the recoil upon them of the columns of blood in the aorta and pulmonary artery.

CONCLUSIONS ON MURMURS ARTIFICIALLY PRODUCED IN THE FOREGOING EXPERIMENTS.

1st. Compression, however slight, of the aorta or pulmonary artery produced a murmur and thrill with the ventricular systole.

2nd. Hooking up a valve of one or both arteries, or perforating a valve, produced a long sighing murmur from regurgitation (Obs. 15 to 18, and 25).

3rd. Passing a bent wire through the walls of the left auricle, so as to hold open the mitral valve, produced a very loud murmur from regurgitation, attended with a strong thrill (Obs. 26).

4th. This wire *did not occasion a murmur when the blood was passing from the auricle into the ventricle*, either during the auricular contraction preceding the ventricular systole, or during the ventricular diastole. Nor was a murmur created at these times in the case of Christian Anderson, though the mitral valve was contracted to the size of the little finger, and the tricuspid to that of the middle finger. I have met with several similar cases, and therefore entertain doubts whether Laennec was correct in stating that a murmur was occasioned by contraction of the auricular valves, during the influx of blood from the auricles: still, I do not absolutely deny this, but I feel assured that the murmur, if it exist at all, is invariably very feeble; and I have reason to know that the *loud* murmur usually ascribed to this source, proceeds from regurgitation through the semilunar valves.

ERRONEOUS OR DEFECTIVE THEORIES OF THE SOUNDS OF THE HEART.

According to M. Raciborski, the muscular "is the oldest theory of the sounds, being the one which appears in the works of Galen, Harvey, Senac, Haller, Bichat, and especially Corvisart, who all ascribed the sounds *to the successive shortening of the muscular fibres. Laennec* embraced the received opinion, which appeared to him sanctioned by the experiments of Wollaston and Erman on bruit musculaire. He thought that the first sound depended on the contraction of the ventricular fibres, and the second on that of the auricular; and when Dr. Barry had demonstrated that the auricles were almost motionless and in a state of permanent plenitude, Laennec had recourse to the contraction of the *appendices* of the auricles to account for the second sound" (Raciborski, du Diagnostic. p. 760). Laennec's theory of the first sound is defective because it overlooks the valvular sound: his theory of the second is wholly incorrect because the auricles contract before the ventricles, because the sound continues when the auricles are motionless (my Exp. p. 24), and because it is demonstrated above that the closure of the semilunar valves is the sole cause of the second sound.

Dr. C. J. B. Williams espoused the muscular theory in his book in 1828. In his second edition he says of the first sound, "In the former edition, I ventured to class it among the muscular sounds which Dr. Wollaston first noticed to occur in all cases of rapid muscular contraction. A good example of it may be obtained on applying a stethoscope to the neck of a person who holds his head back towards the opposite side, and then throws the platysma myoides into contraction" (Rational Exposit. Appendix, p. 199). I shall hereafter show that this is nothing more than a murmur in the jugular veins (see Venous Murmur); yet it unequivocally proves what was Dr. Williams' *type* of a muscular sound. After witnessing my experiments, however, in Nov. 1834 and Feb. 1835, he inconsistently disclaims the type to be his own, for he says "Dr. Hope seems to have taken the dull, rumbling sound described by Dr. Wollaston as the *type* of muscular sound." In short, after my experiments had disclosed the existence of a sound of muscular extension or tension, Dr. Williams adopted a new type of bruit musculaire, but forgot his old one. I have shown that this exclusively muscular theory of the first sound is defective by excluding valvular sound. Dr. Williams' theory of the second sound up to the time when he witnessed my experiments, was, "It is either seated in the parietes of the ventricles rendered momentarily tense by the sudden influx of the blood, or occasioned by the motions of the fluid itself during the diastole" (Rat. Exposit. second edit. Appendix). Incorrect, because my experiments demonstrated that the closure of the semilunar valves was the sole cause of the second sound.

Mr. Turner. Second sound, caused by collapse of the heart on the pericardium during the ventricular diastole. Incorrect, because the sound continues though the pericardium be removed.

Dr. Corrigan. First sound and impulse, caused by the rush of blood into the ventricles, occasioned by the auricular contraction. Incorrect, because the first sound and the impulse continue perfect while the auricles are motionless (my Exp. p. 24). Second sound, caused by collision of the internal surfaces of the ventricle, at the end of the ventricular systole, which systole he supposes to be instantaneous. Incorrect, because fully proved that the ventricular systole, the impulse and the pulse of arteries near the heart, do not coincide with the second sound, but precede it by a very considerable interval (my Exp. p. 17 and 21).

M. Pigeaux. First sound, by the rush of blood into the ventricles during their diastole. Identical with Dr. Corrigan's and incorrect for the same reasons. Second sound, by the collision of the blood against the walls of the aorta and pulmonary artery, during the ventricular systole. Incorrect, because proved that the second sound occurs at a considerable interval after the impulse and pulse in arteries near the heart—consequently, after the ventricular systole. Also disproved by the second sound being annihilated whenever the semilunar valves are disabled (Exps. on the Sounds, p. 33. Obs. 3, 14 to 18).

M. Marc d'Espine. First sound, the pure simple effect of the ventricular systole. Incorrect, as it excludes the sound of the auricular valves. Second sound, the pure simple effect of the ventricular diastole. Incorrect, because proved that the semilunar valves are the sole cause.

Dr. Billing, subsequently *M. Rouanet*, (who derived his idea from Dr. Carswell) and simultaneously *Mr. Bryan*. First sound, by closure of the mitral and tricuspid valves during the ventricular systole. Imperfect, as it wholly excludes muscular sound. Mr. Bryan, however, recognized a sound from muscular tension in one of his papers in 1833, but in his subsequent strictures on the Dublin Committee of August 1835, he ascribes the sound to valvular closure alone. Second sound, by closure of the semilunar valves. Correct.

M. Bouillaud adopts M. Rouanet's theory, but thinks that "the smart recoil of the sigmoid valves against the walls of the aorta, constitutes an element or condition of the first sound which ought not to be neglected;" he thinks also that the recoil of the auricular valves against the walls of the ventricles may contribute to the second sound. These additions are superfluous, if not incorrect.

Mr. H. Carlile. First sound, produced by the rush of blood into the arteries during the ventricular systole. Incorrect, because the sound continues though the orifices of the arteries be obliterated by compression. Second sound, by closure of the semilunar valves. Correct.

M. Majendie. First sound, produced by the collision of the apex of the heart against the ribs during the systole; and the second sound by the collision of its anterior surface against the sternum during the diastole. Incorrect, because both sounds remain perfect when the sternum and ribs are removed.

CHAPTER III.

PHYSIOLOGICAL PHENOMENA OF THE HEART'S ACTION AND SOUNDS, FOUNDED ON THE FOREGOING RESEARCHES.

According to the data supplied by the foregoing experiments and researches, and by the cases appended to this work, the physiological phenomena of the heart's action appear to be as follows:—

I. *The phenomena of the heart's action in the order of their occurrence.*—The first motion of the heart which interrupts the interval of repose, is the auricular systole. It is a slight* and very brief contractile movement, more considerable in the auricular appendix than elsewhere, and propagated with a rapid vermicular motion, towards the ventricle, in the systole of which it terminates rather by continuity of action, than by two successive movements.†

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* I suspect that during palpitation it becomes much more considerable; for, in subsequent experiments on young asses poisoned with woorara, which less impairs the action of the heart, I found the auricular contractions very full and active whenever the pulse rose 20 or 30 beats above the natural standard; but when the palpitation subsided, the contractions again became slight.

† It has been supposed by Laennec, who is supported by Bouillaud, that the auricular systole may produce an impulse. As I believe this opinion to be erroneous, I shall briefly present, and comment upon the data on which it is founded. "If the contraction of the auricles, says Laennec, produces, in some rare cases, a phenomenon analogous to the impulse of the ventricles during their systole, it is easy to distinguish the one from the other. In fact, when the auricular systole is accompanied with a sensible movement, this movement is much more deep: it seems even that the *heart withdraws itself from the ear* . . . In all cases, it is very slightly marked in comparison of the sensation of heaving produced by the contraction of the ventricles when their walls are of a good thickness."

Now, as it has been shown in the foregoing experiments that the contraction of the auricle is a *slight* movement, and that it withdraws the auricular appendices from the walls of the chest instead of causing them to advance forward,—circumstances which render it impossible for the auricles to create an impulse by impinging against those

The ventricular systole commences suddenly, and, at the moment when the auricular valves close, a strong lateral impulse is felt—especially in the vicinity of the auricles, while the walls attain an extreme degree of tense hardness: simultaneously, the apex is tilted up and is drawn towards the base. The systole terminates in the diastole, which is marked by the second sound. Synchronous with the systole are, the first sound, the impulse of the apex against the ribs, and, in vessels near the heart, the pulse; but, in vessels at some distance, as the radial, the pulse follows at a barely appreciable interval.

The systole of the ventricles is followed by their diastole, during which they return, by an instantaneous expansive movement sensible to the touch and sight, to the same state (with respect to size, shape, position, &c.) as during the previous interval of repose. This movement or diastole is accompanied by the second sound, by an influx of blood from the auricles, by a slight retractile motion of these cavities most observable at their sinuses, and by a retrocession of the apex of the heart from the walls of the chest.

Next succeeds the interval of repose, during which the ventri-

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walls, I have no doubt that what M. Laennec noticed, in the "rare cases," alluded to, was, the diastolic impulse of the ventricles, a phenomenon to which attention was first drawn by the writer as a sign of hypertrophy (see Impulse of Simple Hyp.); and I am confirmed in this opinion by his expression that the heart, in giving this impulse, "*seems to withdraw itself from the ear.*" See also my Exp. p. 15.

M. Bouillaud, however, espouses Laennec's opinion. "If the systole of the auricles," says he, "is not accompanied with an appreciable impulse in the healthy state, it is not the same in certain cases of disease." After quoting the above passage from Laennec, he proceeds, "Out of the examples which I could adduce in support of what has just been said, I shall choose the following. In a female affected with an enormous hypertrophy of the heart with induration of the mitral valve, a movement of impulse was distinctly seen to be communicated to the left supra-mammary region, to about an inch below the clavicle (in the 2d and 3d intercostal spaces); the finger placed on this part was repelled, as it were, by a very marked shock. This movement, which could only be attributed to the systole of the dilated and hypertrophous left auricle (for the ventricular beats were felt two inches lower down) *alternated* with another which corresponded to the (auricular) diastole. *This double movement of undulation—of contraction and dilatation, perfectly imitated that presented by the denuded heart*" (Traité I. 149. 1835). As this chosen case was not attended with a post-mortem examination, it does not prove M. Bouillaud's point. For my own part, I have scarcely a doubt that the case was one of dilatation of the pulmonary artery, which I have found to produce the precise symptoms described by M. Bouillaud (see Dilat. of Pulm. Artery).

cle remains at rest, in a state of fulness, though not distention, through the whole period intervening between the second and the first sounds; but the auricle remains at rest during the first portion only of that period, the remainder being occupied by its next contraction, with which recommences the series of actions described.

The rhythm of the heart, that is, the duration of the several parts of this series, which constitute what may be called a beat, is much the same as described by Laennec: viz. 1. The ventricular systole occupies half the time, or thereabouts, of a whole beat. Mr. Bryan says a third only (see p. 20, note). 2. The ventricular diastole occupies a fourth, or at most a third. 3. The interval of ventricular repose occupies a fourth, or rather less, during the latter half of which the auricular systole takes place.

II. *Causes, mechanism, and objects of the motions.* Though Haller accurately noticed the motions of the heart, he was unable to account for the particular order of their occurrence. Hence he says "The reason is a postulatum (postulatur ratio) why, first, the right and simultaneously the left auricle contract, while in the meantime the ventricles rest relaxed; why, a little after, the auricles are relaxed, but the ventricles contract; and then, in a third portion of time, the ventricles *repose* relaxed, but the auricles again smartly contract" (De Motu Cordis; Lugduni Batavorum; 1737, p. 37). The reasons required can now be assigned. The auricles, which are always in a state of fulness though not distention, arrive, from the progressive influx of blood during the first portion of the ventricular repose, at the state of distention, by which they are stimulated to contract. The object for the contraction at this moment, is, to propel a small additional quantity of blood into the ventricles, already full, for the purpose of bringing them from the state of mere fulness to that of distention:—an object which could not be accomplished without a contraction, as the blood could not otherwise force its way into the ventricles against the resistance offered by their elastic parietes. These cavities, then, being brought to the state of distention, are thereby stimulated to contract. They expel a greater or less proportion of their contents—the whole in small animals, frogs for instance,—as is proved by the ventricles becoming pale;

but in large animals, as the ass, they do not *appear*, judging from the diminution of size, to expel the whole, though, as the ventricular walls are opake, whether they do or not, does not admit of demonstration. During the act of expulsion the apex is tilted forwards and upwards and occasions the impulse against the ribs. On the mechanism of this motion, a point hitherto much disputed, it is necessary somewhat to enlarge.

When the heart of an animal, as a frog, rabbit, dog, turtle, &c. is detached from the body before organic life is extinct, and placed upon a table, it continues to act, and each contraction elevates the apex. Hence it is unquestionable that the muscular fibres have an inherent faculty of producing this action. The manner in which the action is accomplished is very visible on inspection. During the state of relaxation, the heart lies collapsed and flattened, with a large extent of its under surface applied to the table; on contracting it starts up, and, assuming a more rounded form, is sustained by a comparatively small point of contact. The apex is, consequently, elevated, and the elevation is greater in consequence of the base, from its superior weight, being the more fixed part. The action is closely analogous in the living subject. Before describing it, I shall advert for a moment to the anatomical disposition of the parts of the heart.

In large animals, as the human species, the auricles, especially the left, are attached to the posterior part of the base, and the aorta and pulmonary artery spring from its anterior part. These vessels are the fixed points towards which the fibres of the heart contract during the ventricular systole, and their stability is increased by the injection and distention which they undergo during the systole. The sinuses of the auricles being constantly full, even during the contraction of the appendices, and regurgitation of their contents into the veins being opposed by the elasticity of the venous coats, by the pressure of the surrounding parts, by the capillary vis-a-tergo, and by the atmospheric pressure, with a power exceeding the weight of the ventricles, the auricles form an almost unyielding fulcrum beneath the ventricles during the systole of the latter.

Such being the anatomical and physiological state of the parts, during the ventricular systole the braced fibres, contracting towards the aorta and pulmonary artery in front, draw the tense and rounded body of the ventricles upon the auricular sinuses behind.* Consequently, the apex of the ventricles is tilted up; and this motion is performed with considerable velocity, because, if I may be allowed the illustration, the apex is the long arm of the lever, the auricles being the fulcrum, and the moving power at the aorta and pulmonary artery. In proportion as the ventricles contract to their extreme, the apex is not only retracted towards the base, but thrown more and more forward by the auricular distention, advancing in the same progression to its extreme. Another circumstance probably contributes to the elevation of the apex; namely, the retropulsion of the auricular valves: for, as these act on a column of fluid which offers a resistance greater than the weight of the heart, the action is reflected on the organ itself, and impels it forward. I have seen the impulse of the heart prodigiously increased by an aneurism of the aorta forming a solid fulcrum immediately behind the heart: also by adhesion of the pericardium binding the organ in front of the spine: by pleuritic effusion throwing it into the same position: and Dr. Stokes says, by tubercular consolidation of the lung behind the heart. All these causes act by affording a more unyielding fulcrum behind the organ than the healthy lung, and they constitute pathological corroborations of the view of the heart's impulse which I maintain.

The diastole appears to be occasioned by several concurrent causes; viz. 1. That power of the muscle (whether elasticity, or something more, is unimportant) by which it reverts from the state of contraction to that of relaxation, and in virtue of which it exercises a degree of suction. 2. The distention of the auricles, which is greater at the moment of the diastole than at any other, as they have been filling during a longer period—namely, that of the ventricular contraction or about half of a whole beat. 3. The weight of the ventricles collapsing from their systole on the distended auricles beneath them. 4. The width of the auriculo-ventricular orifice, which allows the blood to shoot in without impediment. It is manifest that as so many powerful causes conspire to effect the influx of the blood, an auricular contraction at this time, and for this purpose, as imagined by Laennec, would be superfluous. The draught of blood from the auricles during the diastole, causes the slight retraction of these cavities observable at that moment.

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* Mr. Bryan has shrewdly observed upon this that it "implies imperfection in the mechanism of the heart; for if, when the ventricles contract, they make pressure on the auricles, the ventricles must waste a portion of their power in impeding the action of the auricles" (Lancet, vol. xxiv. p. 783). But it is stated above that the auricles contain a column of fluid which offers a resistance greater than the weight of the heart—therefore the action of the auricles would not be impeded by that weight. It is not improbable that the pressure of the ventricles during their systole operates as a salutary check on the auricles, preventing them from becoming immoderately distended during the long period of the ventricular systole.

The object of the interval of repose, is, to afford rest to the organ, and no arrangements could answer this object so completely as those described. We see that the ventricles, by their diastole, are brought to the state most favourable to their repose —that of natural fulness without distention, and in this state they remain, to employ an approximative calculation very nearly exact, one fourth of each beat or six hours in twenty-four; but, if we admit that the diastole also is a kind of repose to the ventricles, we must add another fourth, making the whole period half a beat or twelve hours, which is the period assigned by Laennec (De l'Auscult. tom. ii. p. 408). Had the auricular systole succeeded the ventricular and immediately brought the ventricles from the state of contraction to that of distention, as supposed by Laennec, there could have been no repose, as distention implies the exertion of the tonic power of a muscle, which, as is well known to those who have to reduce dislocated joints, is eminently productive of fatigue. Nor, indeed, could any *interval* of action then have taken place; for, admitting distention to be the stimulus of the ventricles, their contraction would have ensued instantly on their being brought into the state of distention. We see, further, that according to the theory which I advocate, the auricles, by evacuating their contents into the ventricles at twice instead of once, avoid the excessive distention at which they must have arrived had the blood been accumulating in them during three fourths of a beat. Although their systole occupies only about one eighth of a beat, or half the time between the second and first sounds, the whole of the remaining seven eights is not devoted to repose; for, during the greater part of this time, the auricles are in a state of greater or less distention, which, as above stated, is not repose. Under these circumstances it is too hypothetical, if not impossible, to estimate by numerical calculation the exact amount of the auricular repose, but analogy, countenanced perhaps by a rude calculation, leads us to infer that it must be about equal to the ventricular. Laennec estimates it at one half more, or eighteen hours in twenty-four, but this he does on the assumption that the auricular contraction occupies one fourth of a beat, and without allowing for the fatigue occasioned by the state of distention into which the auricles are brought during a portion of the intervals between their contractions.

As M. Majendie has adopted a kind of *alternate* theory of the heart's movements, which has been incautiously copied, on the authority of so high a name, by the bulk of systematic writers, it may be well to point out its inaccuracy. "If," says he, "the heart of a *living* animal is denuded, we easily see that the auricles and ventricles contract and dilate alternately. These movements are so arranged, that the contraction of the auricles takes place simultaneously with the dilatation of the ventricles, and, *vice versâ*, that the contraction of the ventricles coincides with the dilatation of the auricles" (Quoted by Bouillaud, Traité I. p. 87). The great defect of this view is, that it leaves no interval of repose. It is easy to see how M. Majendie has been misled, namely, by operating on *living* animals; for I have always found, that when the animal unfortunately retained or regained the slightest degree of sensibility, the action of the heart was so violent, convulsive, and rapid, as to present the appearance of alternate action described by M. Majendie. In small animals, also, as rabbits, whose pulse beats 150 to 200 a minute, the same appearance is generally presented even though they have been completely killed; for the interval of repose is too brief to be distinctly appreciated by the eye. Nay, in asses poisoned by woorara, much the same appearance is presented whenever the pulse is accelerated twenty or thirty beats above its natural standard of forty or fifty, the contraction of the auricle then becoming more active and extensive, and encroaching so much on the interval of repose as to render it indistinct to an unpractised eye. I am, therefore, inclined to think, that during palpitation or naturally accelerated action, the period of repose actually is encroached upon. The muscle, indeed, can bear an occasional and temporary encroachment of this kind with impunity; but when palpitation is long continued, we know that it issues in hypertrophy, or dilatation, or both.

The ocular deception under which M. Majendie has laboured is corroborated by an appeal to physiological principles. Let us inquire how the heart would act, on his alternate principle, in large animals with a pulse of fifty, as in many human subjects, or of forty or less, as in horses, &c.; or let us take for illustration a still more striking case of a gentleman, (whom I at present see in common with several other practitioners,) with a pulse of twenty-eight, without the slightest intermediate beat or sound of the heart. The second sound follows the first almost as quickly as when the pulse beats sixty or seventy: consequently there is a period of repose of about a second and a quarter, as three quarters of a second suffice for an ordinary systole and diastole. What, then, on M. Majendie's view, must be the state of the heart during this second and a quarter of repose? The ventricles *must* be in the state of diastole, because this state follows the second sound. Now the ventricular diastole, says Majendie, is synchronous with the auricular systole: consequently, the auricles, after their systole, must remain in the state of spasmodic constriction for the period of a second and a quarter waiting for the next contraction of the ventricles, which are unexcited by the stimulus of distention! This is monstrous! Its physiological impossibility is palpable. How much more simple and natural and beautiful to suppose that the relaxing ventricles refil, without distending themselves, from the auricles, and that the whole organ then remains in repose till the progressive venous influx provokes the next auricular contraction! How admirable the arrangement by which the auricles tranquilly deliver their blood at twice, instead of the single, violent, alternate delivery supposed by the view of M. Majendie! Fortunately, his high authority is opposed by that of Harvey and Haller, not to mention the experiments of the writer, and the repetitions and confirmations of them by the Dublin and London Committees of the British Association. M. Bouillaud appears to follow Majendie (Traité de Bouillaud I. p. 136, 1835); and this error has betrayed him into several others respecting physical signs. Dr. Bostock also follows

Majendie, but evidently from inadvertence; for he applauds Haller's experiments, which coincide with the writer's, and are opposed to the alternate theory of Majendie.

III. *The Causes and Mechanism of the Sounds.* This subject is so fully discussed in the last section (see Conclusions, p. 39) that a brief summary will here be sufficient.

First Sound. This is compound, consisting, first, of the click of the auricular valves: secondly, of the sound of muscular extension—a loud, smart sound, produced by the abstract act of sudden jerking extension of the braced muscular walls at the moment when the auricular valves close: thirdly, of a prolongation, and possibly an augmentation, by *bruit masculaire,* i. e. the dull, rumbling sound of ordinary muscular contraction.

The valvular click gives smartness and intensity to the commencement of the first sound, and in feeble hearts, in which the sound of extension and of *bruit musculaire* are absent, the click alone is heard, causing the first sound to be identical in quality with the second. This occurs, for instance, in dilatation with attenuation. The sound of muscular extension superadds bluntness and loudness to the valvular click, and is probably a principal cause of the extraordinary intensity of the first sound, often observed in violent palpitation. It differs from the sound of costal percussion with metallic cliquetis, which imparts a double character to the first sound and only exists under the circumstances described at p. 41. The *bruit musculaire* forms a gradually diminishing prolongation of the sound to the end of the act of contraction: but when the heart acts feebly, either from disease, or from mere temporary exhaustion or faintness, the *bruit musculaire* may be partially or wholly absent.

Second Sound. This results from the sudden expansion of the semilunar valves, occasioned by the reflux upon them of the columns of blood in the aorta and pulmonary artery during the ventricular diastole.

The auricles do not contribute to the production of either of the sounds; as, in the experiments on the ass, they were heard in equal perfection when the auricles were in a state of immobility. Nor does the auricular contraction, in my opinion, produce any sound whatever; for the movement during tranquil action of the heart, in large animals at least, is too inconsiderable to be capable of it. Further, there are no circumstances of structure or resistance to occasion valvular or muscular extension. Finally, no third sound of the heart is ever heard. M. Bouillaud, indeed, has pronounced this last assertion to be inaccurate; and, in proof, he cites "cases of disease of the heart in which he had heard, and caused a great number of persons to hear, three, or even four sounds instead of two during a single rhythm." These supernumerary sounds he ascribes to auricular contractions, but he labours under an entire misapprehension. The truth is, that the sounds really result from ventricular contractions, but contractions so feeble as not always to produce a pulse perceptible at the wrist. The proof is, that, in almost all such cases, the third and fourth sounds are every now and then attended with a barely perceptible pulse, which inevitably connects the sounds with ventricular contractions. Sometimes the pulse can be felt in the carotid when it is imperceptible in the radial. These cases are far from uncommon. I have notes of twenty or thirty, and my limits alone prevent me from transcribing several before me.

The first sound is best heard at that part of the præcordial region where there is dulness on percussion from the heart being in contact with the chest; for the sound is best conducted to the surface through a solid medium, without the intervention of the spongy, ill-conducting tissue of the lungs. The second sound is best heard over the semilunar valves, viz. on the sternum, opposite to the inferior margin of the 3d rib, and thence for about two inches upwards, along the diverging courses of the aorta and pulmonary artery respectively, the sound *high up* the aorta, proceeding mainly from the aortic valves, and that *high up* the pulmonary artery being mainly from the pulmonic. This subject is more fully developed at p. 3. It has not been fully understood by other authors, nor was it by the writer in the first edition of this work.*

For an account of the extent over which the healthy sounds are audible and the circumstances which occasion variations, the reader is referred to *Dilatation*, sounds of; and also to Hypertrophy.

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* The author of the Rational Exposit. has criticised that edition, but seems to have forgotten that I supplied him with the criticisms. My rectifications were published previous to his criticisms in the appendix to the 2d edit. of the present work.

First principle of the heart's motion. After having studied all the physiological phenomena of the heart's action, an ulterior question naturally presents itself—what is the first principle,—the primary spring, which gives motion to the great organ of the circulation. As this is rather a question of physiological interest, than one, the determination of which is essential to the present subject, I shall merely glance at the existing opinions, and leave the reader to prosecute the inquiry by referring to original sources of information.

It is the persuasion of many distinguished physiologists, particularly those of the French school, founded, as they conceive, on experiment and observation, that the nerves of the heart constitute its motive principle. But, as these nerves are derived from two sources,—the cerebro-spinal, and the ganglionic systems, it was a question which of the two were destined to impart the faculty of motion. Le Gallois ascribed this faculty to the spinal nerves; but his conclusions were subverted by the researches of Lallemand, who found that the heart beat in the fœtus though destitute of spinal marrow; and by the experiments of Wilson Philip, Mayo, Clift, and many others, who found that the action of the heart survived the destruction of the spinal marrow, and even the excision of the organ out of the body. Hence it resulted that, while the cerebro-spinal nerves or par vagum (according to the brilliant discoveries of Sir Charles Bell and the recent extension of them under the name of the excito-motory system discovered by Prochasca), connected the heart with the lungs, the stomach, the thoracic muscles, the face,—with all the parts, in short, associated in the functions of respiration and expression, and, in fact, with the whole system; the ganglionic nerves, or, in other words, the great sympathetic, was the principle which imparted the faculty of motion. Thus it was explained how the action of the heart was independent of the will, while it was strongly under the empire of the passions and of corporeal nervous sympathies.

Mr. Mayo, on the contrary, founding his opinion on a train of profound and ingenious reasoning, partly developed in his work on physiology, but which he has done me the favour to explain more at length, entertains the belief that the motive principle of the heart is an innate power independent of the nerves; and that, while it is the *natural state* of voluntary muscles, both in the living body and before the loss of irritability after death, to remain relaxed, unless excited by special impressions; it is, on the other hand, the *natural state* of the heart, an involuntary muscle, under the same circumstances, to contract and dilate alternately for a time, in the absence of external impressions. In the turtle, an extremely vivacious animal, the alternate actions continue for a very long period. I have seen them last for upwards of an hour, though sections were made both longitudinally and transversely into the cavity of the ventricle. The motive principle, whatever it be, appears to be more or less exhausted by each contraction; for a puncture, made immediately after the effort, does not cause a repetition of it; but, made at the interval of a few seconds, it produces the effect.

Whether the motive principle be nervous, or an innate power, it is sufficient for our present purpose that the organ replies to a stimulus. This stimulus, in the natural state, is the blood; and by flowing into the heart in suitable quantities at definite intervals, according to the principles above explained, it appears to maintain the action of the organ in a state of regularity. Mr. Granger, advocating the existence of the reflex function in the ganglions of the great sympathetic, maintains the same view. "The heart," says he, "may be selected for the sake of illustration; the blood reaching the cavities of that organ makes an impression upon the *incident* branches of the cardiac nerves (i. e. those which go *to* the cardiac ganglion); this impression excites the *power* of the cardiac ganglion, the influence of which being transmitted by the *reflex* cardiac twigs (i. e. those going *from* the ganglion), causes the muscular substance to contract." He adds in a note, "If the cardiac nerves and ganglion are essential to the heart's action—and that they are so, will, I feel confident, be ultimately established—their action *must* be 'excited' (by the blood stimulating the *incident* branches); or otherwise we must allow that the ganglions can spontaneously stimulate the muscular fibre, or, in other words, that they are intelligent agents" (On the Spinal Cord, p. 142, 1837).

CHAPTER IV.

PATHOLOGICAL PHENOMENA OF THE HEART'S ACTION AND SOUNDS.

SECTION I.

MODIFICATIONS OF THE MOTIONS AND SOUNDS BY HYPERTROPHY, AND DILATATION.

BY *Simple Hypertrophy*, the impulse is increased and the sounds are diminished. "The impulse," says Laennec, "is ordinarily sufficiently strong to heave the head of the observer in a very sensible manner, and sometimes it is so strong as to produce a shock disagreeable to the ear. The greater the hypertrophy, *the more time that heaving takes for its performance*, and, when the disease is carried to a high degree, we evidently perceive that it takes place by a *gradual progression;* it seems as if the heart swelled and applied itself to the walls of the chest, at first by a single point, then by its whole surface, and, in the next place, suddenly sunk back" (s'affaisse). In considerable hypertrophy, and still more in hypertrophy with dilatation, this sinking back terminates in a jog or shock, to which I called attention in the first edition of this work, as a new sign of these affections, under the name of *back-stroke*. The term *diastolic impulse*, however, is more descriptive, and I shall therefore employ it.

The *first sound*, i. e. that attending the ventricular systole, is duller and more prolonged than natural, in proportion as the hypertrophy is more considerable; and, when this exists in an extreme degree, the sound becomes nearly extinct, but never, according to my observation, wholly so, as stated by Laennec. The *second sound*, i. e. that produced by the closure of the sigmoid valves during the ventricular diastole, is weaker than natural: Laennec says that in extreme cases it is scarcely perceptible; but I have always found it distinct when the stethoscope was placed accurately about an inch or two higher up than the sigmoid valves.

The causes of these modifications are very intelligible. The power of the impulse is increased in the direct ratio of the hypertrophy; and the movement is a progressive heaving, because the hypertrophous ventricle, from being thick and unwieldy, contracts slowly and with a gradual progression. For the same reason the *first sound* is diminished,—is dull and stifled; because, as the closure of the auricular valves is sluggish, it is attended with a less jerk of extension both of the valves and chordæ tendineæ and of the ventricular walls. I think also that the sound is somewhat deadened by the increased thickness of the ventricular walls through which it has to be transmitted.

The *second sound* is also diminished, because the ventricular diastole, no less than the systole, being performed more sluggishly, the recoil of the blood on the sigmoid valves is less smart; and this smartness is still farther diminished in hypertrophy with contraction, because the quantity of blood expelled by the ventricles is insufficient adequately to distend the arteries.

By *Simple Dilatation*, and *Dilatation with Attenuation*, the impulse is diminished, often to the extent of being imperceptible. When perceptible, it is a sudden brief blow, which communicates a shock or vibration to the thoracic walls, but has not power or duration to elevate them. The reason is, that, as a thin muscle has less power, but greater facility and rapidity of motion, than a thick one, the attenuated ventricles contract on their contents with greater velocity than natural, but their action is more feeble: accordingly, the impulse is diminished, and its power is sooner exhausted,—whence the brevity of the shock. The apex, in other words, is suddenly tilted forwards, and its force seems to be expended, as it were, in the act.

The first sound in dilatation, becomes loud, brief, and clear, like the second. This arises from the muscle, in consequence of its thinness, contracting with increased facility and velocity,—

whence the extension of the auricular valves with their chordæ tendineæ, and of the muscular walls themselves, is more sudden and smart. The sound is not prolonged by *bruit musculaire*, apparently in consequence of the feebleness of the contraction. In dilatation with attenuation, the first sound is so brief and often feeble a click, that I believe it to be produced by valvular extension alone.

The second sound is more or less increased, because the thin ventricle, from having greater facility of movement, performs its diastole, as well as its systole, with greater velocity; whence the recoil of the sigmoid valves is more sudden. In dilatation with extreme debility of the organ, however, I have often found both sounds weaker than natural, from the excessive feebleness of the heart's action.

By *Hypertrophy with Dilatation.* The modifications occasioned by this affection are compounds of those of hypertrophy and those of dilatation. The contractions of the ventricles give a strong impulse—"abrupt, dead (sec), violent blows, which strongly repel the hand" (Laennec de l'Auscult. tom. ii. p. 515): they partake, in short, of the power of hypertrophy and the smartness of dilatation. *The first sound* is increased, sometimes exceedingly, so as, according to my observation, to be louder than in any other disease of the heart. This is in consequence of the violence and velocity of the valvular and muscular extension. The sound is prolonged by *bruit musculaire*. *The second sound* is increased to its maximum, partly from the ventricular diastole being quick and vigorous, but partly also from the tension of the arteries being increased by the preternatural quantity of blood injected into them, whence the recoil of the blood on the sigmoid valves is more violent and rapid.

Hypertrophy with dilatation is occasionally accompanied, especially during palpitation, with a soft and slight bellows-murmur, an exposition of the cause of which I reserve for a future section on *Murmurs from Hypertrophy with Dilatation.*

The impulse and sounds, in any affection of the heart, may partially, and the impulse even totally fail, when the organ, either from its own debility, or an obstacle in the course of the circulation, is gorged with an accumulation of blood which exceeds its propulsive power; and also when the vital powers are reduced by any cause whatever. This is not only indicated by pathology, but is demonstrable on the stupified living animal; for, if artificial respiration be temporarily suspended, the diminution of sound and impulse immediately takes place, and it may thus be produced and removed at pleasure. The heart, during the intervals of inflation, is seen in a gorged state, scarcely contracting or dilating (See Experiments, p. 23).

SECTION II.

MURMURS PRODUCED BY VALVULAR DISEASE.

By valvular disease the sounds acquire various morbid murmurs, as those of bellows, sawing, filing, rasping, whistling or a perfect musical tone; and these sounds are valuable signs of valvular disease. I shall first notice the circumstances under which they occur, and subsequently advert to the mechanism of their formation, and explain the causes of their varieties. It will be seen that they perfectly assimilate with, and substantiate, the view that I have taken of the motions and sounds of the heart.

The illustrious author of Auscultation was acquainted with two circumstances only in which valvular murmurs were heard. "The bellows-murmur," says he, "attends the contraction of the left auricle (by which must now be understood the ventricular diastole or second sound) when the mitral valve is affected, and that of the ventricle when the induration affects the sigmoid valves of the aorta." He does not expressly say whether he intends these signs to apply equally to the valves of the right side, but the following general statement will perhaps admit of that construction:—"Bellows-murmur exists almost constantly in the orifices of the heart in individuals affected with contraction of the orifices of that organ" (ii. 441).

M. M. Bertin and Bouillaud, in 1824, adopted, without extending, the valvular murmurs of Laennec, but they distinctly apply them to the right, as well as to the left side (Traité, p. 225).

Neither these authors, nor Laennec, nor any French writer was acquainted with murmurs from regurgitation through the several valves; and as they necessarily confounded these with the other murmurs, I have not the slightest doubt that it was this circumstance, as well as Laennec's erroneous idea that murmurs might result from mere spasmodic contraction of the heart and arteries (Traité II. 440), which caused this acute observer to contradict himself in his second edition by saying "bellows-murmur does not suppose any organic lesion in the heart and arteries" (Traité II. 443).

To the murmurs of Laennec, I added, in the first edition of this work in December 1831, the murmurs from regurgitation, thus assigning to each valve a double murmur—one, from the blood flowing in the natural direction; the other, from its flowing retrograde when the valve was permanently patescent.* I at the same time controverted the murmur from spasm of Laennec,† and showed by the experiments and arguments offered under the subjoined head of *murmurs of the heart and arteries independent of organic disease*, that the murmurs did not depend on spasm, but on other very appreciable causes; and that they were easily distinguishable from the murmurs of valvular disease. Eight years of additional experience have confirmed me in the general accuracy of the views which I then took; but a fuller and more precise knowledge of all the circumstances now enables me to correct a few minor errors, and to make such additions as will, I hope, render the diagnosis of valvular disease not only the most certain connected with the whole subject, but so simple and easy as to be readily attainable by the meanest capacities.

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* Dr. Elliotson, I find, published before myself the fact that permanent patency of a cardiac opening was a source of bellows-sound. He candidly adds, "I heard it first from Dr. James Johnson. *Who originally suspected it, I cannot say.* Dr. Johnson imagined he had learned it from Laennec and other writers upon auscultation; but I have found no other notice of it than the erroneous view of Bertin" (Lumleyan Lect. p. 20, 1830). I believe I can explain this. Dr. Johnson probably learned it from his son, to whom I had communicated it; for in 1829 and 1830, he and I studied auscultation together in St. George's Hospital, and I was in the habit of pointing out the regurgitations as a discovery of my own, made in June 1825, in the remarkable case of Christian Anderson, for which, see Index to the cases. I also taught the regurgitations at St. Bartholomew's Hospital in 1826, and at La Charité, Paris, in 1827.

Dr. Corrigan, not aware of Dr. Elliotson's publication or my own, subsequently published a paper in the Edin. Med. and Surg. Jour. No. 111. for 1832, "On a *new* disease of the heart: viz. permanent patency of the mouth of the aorta."

† Dr. Corrigan, I find, had previously done the same in the Lancet of 1829, of which I was not aware. More recently, in 1835, M. Bouillaud has followed in the same track. This doctrine, in short, is now universally exploded.

The circumstances under which I have found murmurs produced in the several valves respectively, are as follows.

Aortic Valves. 1. Systolic murmur. I have found a murmur attend the ventricular systole in every degree of fibrous, fibro-cartilaginous, steatomatous and osseous disease of the aortic valves sufficient to *contract the aperture.* The same remark applies to vegetation on the valves or in the orifice. I have even found considerable murmur produced by mere osseous or even steatomatous asperity of the valves, without contraction—at least, such as could be appreciated: a fact which is easily explained, since it is known that mere roughness, by increasing friction, will produce sound. I have also found murmur created when, without contraction or roughness of the valve or orifice, the aorta immediately above the valves was dilated either in its whole circumference, or partially so as to form a pouch. Here the stream is broken by the divergence of the blood, just in the same way as when it passes from a contracted orifice into a natural-sized aorta. I have found a very loud bellows-murmur produced by an opening, admitting the index finger, from the right ventricle into the mouth of the left ventricle and the aorta (case of Collins); and I have noticed the same murmur in four other cases of malformation with Cyanosis, in which I had not the opportunity of post-mortem inspection. The murmur discovered by Dr. Latham as attending pericarditis, and which he communicated to me in 1826, I soon ascertained to proceed in many cases from the interior of the heart; as I found it continue after the pericarditis had ceased, or the pericardium become adherent. I therefore expressed my belief, in the first edition of this work, that the systolic murmur "might, in some instances, originate partly on constriction of the arterial orifices consequent on inflammation of the lining membrane. For as this membrane is more liable to inflammation where it constitutes the valves, than elsewhere, it is consistent with analogy to suppose that, by its intumescence and loss of elasticity, the orifices will undergo the constriction alluded to. The murmur accompanying the second sound, I am inclined to attribute perhaps entirely to the same constriction, affecting the auriculo-ventricular orifices" or (I should have added) occasioning patency of the sigmoid valves. The accuracy of this opinion has been fully substantiated by my subsequent experience in a great number of cases, and by the researches of Dr. Elliotson, Dr. Watson, Dr. Stokes, and M. Bouillaud.*

Concretions of blood in the heart, if formed before death (of which adhesion and organisation are the best anatomical criteria), may occasion murmurs, either by obstructing an orifice or preventing a valve from closing. They occur principally in acute endo-carditis. I state the fact mainly on the authority of M. Bouillaud, as I do not happen to have lost a patient with acute inflammation of the heart during the last eight years, nor have I noticed the murmur in ordinary cases of polypus before death. Others, however, have. Dr. Elliotson describes a case in his Lumleyan Lectures, p. 18. On the whole, I should think murmur from this cause very rare; and I should imagine that a polypus would be more apt to entangle, and create a murmur in the auricular valves, than in the semilunar.

2. Diastolic murmur of the aortic valves or from regurgitation. I have known this to be occasioned by all the varieties of fibrous, fibro-cartilaginous, steatomatous and osseous disease, and also by inflammatory tumefaction in acute and chronic endo-carditis—one of the most frequent causes of regurgitation. In fact, if any of these diseases contract, or otherwise deform, one or more of the valves, so as to prevent complete occlusion of the orifice, the murmur is produced. I have seen it result from the angles of the valves being detached from their insertions by steatomatous disease (case of Copas); also from a tear of a valve near its angle (Milton and Figs. 10 and 11). I have no doubt that it may also occur from atrophy of the valves producing perforations, though I do not happen to possess an unequivocal case. I have once seen a canal from steatomatous disease, admitting the little finger and half an inch long, pass under the base of an aortic valve and the lining membrane of the heart into the left ventricle; and, though I did not see the patient before death, I have no doubt, from the jerking pulse, that there was regurgitation and therefore

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* When I published the above passage in the 1st edition, I was under the impression that the fact was new. I find, however, that Dr. Elliotson had preceded me in its publication, in his Lumleyan Lectures, which I had not had the good fortune to see. M. Bouillaud in 1835 has mis-stated my opinion, though so distinctly expressed in the above quotation. He says "M. Hope has, at least, had a *glimpse* (a entrevu) of the influence of endo-carditis in producing bellows-murmur," and adds that I restrict it to the diastole !

a diastolic murmur (case of Brown). I have once seen regurgitation and murmur from an aneurism of the aorta immediately above the valves, opening into the right ventricle immediately below its valves (Mitchell, Fig. 21). Though this does not strictly come under the head of disease of the valves, yet it is convenient to notice it here as a possible source of fallacy. On the same principle I may add that, in the case of Evans, regurgitation with murmur resulted from two perforations out of the ascending aorta into the pulmonary artery. Finally, I have seen regurgitation with murmur from mere enlargement of the aortic orifice, whence the valves, otherwise sound, were incapable of closing it (case of R. S., Esq.). Figs. 6, 13, 14, 15, 16, 17, 18 and 20, are of the aortic valves.

I have noticed three circumstances characteristic of aortic regurgitation, to which, after examining a great number of cases, I have not yet met with an exception. 1. The murmur is soft like bellows-murmur, or still more like gently sucking in air through the lips only moderately closed, as in pronouncing the word *awe:* and it is weak, as compared with the much greater intensity which systolic murmurs may attain in the same situation. In the first edition, I explained this weakness as follows:—"I have never found it (the diastolic murmur) strong, and I doubt whether it can be so, as the instantaneous manner in which the ventricle is refilled by its diastole, must prevent the regurgitation from being considerable" (p. 341). Another conspiring cause, however, may be added; namely, that the aortic retrograde pressure is inferior to that of the left ventricle, and therefore it could not retropel the blood with equal force and velocity. 2. The murmur is generally very prolonged—a long sigh, tailing, as it were, the second sound and often extending completely to the next ventricular systolic sound: in one case (W. . . . . Esq.) I even found it prolonged completely through intermissions of the heart's beats. This prolongation I ascribe to the pressure in the aorta being continuous or incessant, and to there being nothing to interrupt it but the next ventricular contraction. 3. The murmur is more audible than a systolic murmur is *below* the sigmoid valves and down the ventricle, though, notwithstanding, it decreases as it descends. This obviously proceeds from the current setting into, and exciting sonorous vibrations within, the cavity of the ventricle. The circumstance is of importance in a diagnostic point of view; for it might create the erroneous belief that the murmur was seated in the mitral, instead of the aortic valves (as both occur during the diastole), unless the auscultator were careful to ascertain that it was loudest at, or above the aortic valves, where a mitral diastolic murmur, always feeble, would be wholly inaudible.

The murmur of aortic regurgitation is of very frequent occurrence, though it is commonly supposed to be rare: the reason of which is, that, before the discovery of the regurgitations (with which many are still but little acquainted), it was necessarily and invariably mistaken for a murmur with the second sound from contraction of the mitral valve. I habitually made this mistake myself before 1825, when I first noticed the regurgitations, and I distinctly see the same mistake in two or three of Dr. Elliotson's cases published in his Lumleyan Lectures in 1830—cases probably taken before he had heard of the regurgitations in the manner above described. He says, for instance, that the murmur was of a "sucking or aspiring" character, that it was "slower," and that it was loudest at the upper part of the heart. In conclusion, I have traced aortic regurgitation and murmur to acute rheumatic endo-carditis far more frequently than to any other cause.

Pulmonic Valves. 1. Systolic murmur. I have never once met with, and ascertained after death, such disease of the pulmonic valves themselves as created a murmur during life. Others, however, have, though very rarely; for, according to Dr. Clendinning's observations on 100 cases, with which my own very nearly coincide, the total proportion of valvular disease on the right side of the heart is only about 1 in 16. I have seen two or three cases, without autopsies, in which I believed the valves to be diseased; but they were principally cases of cyanosis, in which the valvular disease is usually congenital. I have also once seen the orifice of the right ventricle contracted to the size of a quill an inch below the valves, in a case of cyanosis with an opening out of the right into the left ventricle (Collins). Here, the systolic murmur proceeded from both of the morbid apertures. I have once seen an aneurism of the origin of the aorta bulge into the mouth of the right ventricle and contribute to a systolic murmur and thrill over the part (case of Mitchell and Fig. 21).

Dr. Elliotson describes two cases in which lumps of cartilage in the pericardium pressed upon, and contracted the pulmonary artery, so as to create a murmur. I have once met with a case, probably unique, of extensive ossification of the trunks of the pulmonary artery within the lungs, which produced a systolic murmur (Lady R.). I mention this as a source of fallacy, though it does not properly come under the head of valvular disease. Dilatation of the pulmonary artery is another source of fallacy, which will be noticed under its proper head (See Dil. of Pulm. Artery, and cases of Weatherly and L. P.).

Thus it would appear that the majority of cases of systolic murmur in the pulmonic orifice, are connected with lesions, not of the valves themselves, but of contiguous parts.

2. Diastolic murmur of the pulmonic valves. I believe this to be exceedingly rare from disease of the valves themselves, as I have never met with a case, or been able to find one recorded. In Mitchell, the origins of the valves were stretched and separated by the aortic aneurism, so as probably to admit of regurgitation and murmur (see Fig. 21). In Weatherly, the pulmonic orifice was greatly dilated, yet the valves must have closed it, as there was no diastolic murmur. I created this murmur artificially in an ass poisoned with woorara, by making a perforation through one valve (see p. 37-8). We found the murmur soft, prolonged, and audible down the ventricle, exactly as in aortic regurgitation. In the human subject the pulmonic, would probably be louder than the aortic diastolic murmur, because its seat is nearer the surface.

From a rude numerical calculation deduced from the cases that I have seen, I should think that there would be at least thirty chances to one against a murmur connected with the semilunar valves, being seated in the pulmonic set.

Mitral Valve. 1. Systolic murmur, that is, from regurgitation. It was the existence of this murmur in Christian Anderson, who had no disease of the semilunar valves, that led me to the detection of regurgitations in general in June 1825. Since then, I have met with the murmur from every variety and degree of fibrous, fibro-cartilaginous, and osseous disease of the mitral valve and chordæ tendineæ capable of holding the valve permanently open: also, from vegetations. Dr. Elliotson met with it from an organised polypus. It cannot be too strongly inculcated that a slight patency of the valve admitting of regurgitation, may result from a structural lesion not sufficient to present an obstacle to the blood flowing in its natural direction from the auricle into the ventricle. Thus, I feel certain that there is no one lesion which more frequently produces regurgitation than shortening, usually with thickening, of the chordæ tendineæ (e. g. case of Dennis); yet how constantly do we see this lesion completely overlooked, and the valve pronounced capable of discharging its function, because it will allow two or three fingers to pass through it! Another, though less common lesion, apt to be overlooked, is, adhesion of one or both divisions of the valve, especially the posterior, to the walls of the ventricle,—a result of inflammation. Dr. Elliotson gives cases of this in his Lumleyan Lectures in 1830. M. Bouillaud, in 1835, treats of it more fully as a previously unknown lesion (Traité, II. 183). A still more rare source of regurgitation, is, atrophy of the valves rendering them morbidly thin and small, and sometimes attended with cribriform perforation of the membranes. Attention has been drawn to this by Dr. Kingston, in the Medico-Chirurg. Trans. See Figs. 5, 7, 12 and 15.

I have also met with regurgitation and murmur from another circumstance apt to be overlooked; namely, dilatation of the orifice consequent on dilatation of the ventricle, rendering the valve, otherwise healthy, incapable of closing it. I witnessed a striking instance of this in a valuable horse which Mr. Field requested me to see. I found the murmur of mitral regurgitation, and, from the weakness and irregularity of the pulse, judged the reflux to be extreme. The animal died on the following day, and Mr. Field pronounced the mitral orifice to be double its natural size and the valve totally incapable of closing it. The same conditions appear to have existed in the case of H . . .y, Esq., as the murmur ceased when he recovered from the dilatation of the heart.

The murmur of mitral regurgitation is loud, considering the depth of its seat, because it is occasioned by the great force of the ventricular contraction. When, however, that force is much diminished by Softening or by Dilatation with Attenuation, the murmur may be much more feeble—nay, sometimes even extinct. I have, for instance, met with several cases, in which a murmur attended every strong contraction of the ventricle, while the two or three following contractions, so feeble as barely to occasion a pulse, were productive of a valvular click only, without murmur. In another case (Mrs. ——l——n) in which the left ventricle was dilated, attenuated to one third of an inch, and greatly softened, and the mitral valve contracted into a slit which only admitted a writing-quill, no mitral murmur attended either the systole or diastole.

Of all murmurs, that from mitral regurgitation is, according to my observation, the most frequent. Dr. Elliotson, in 1830, said it was rare, and that aortic systolic murmurs exceeded all others in frequency (Lumleyan Lectures, p. 22): but at the period to which his remarks refer, he appears to have been practically unacquainted with the regurgitations, and therefore included mitral regurgitations under the head of aortic murmurs. M. Bouillaud, in his work in 1835, seems equally disposed to give the numerical predominance to aortic systolic murmurs, and apparently for the same reason; for, from his avowed inability to distinguish the particular seat of valvular disease, and from his remark that he believed M. Filhos had attached too much importance to regurgitations, it is evident that he could not have had much practical knowledge of them at that period.

2. Diastolic murmur of the mitral valve. Any lesion of the valve capable of sufficiently contracting its aperture may, under the limitations presently to be described, give rise to this murmur. It was one of the only two murmurs discovered by Laennec, and was long supposed to be of frequent occurrence. But I believe that this opinion is incorrect, and that it has resulted from the murmur having been confounded with that from aortic regurgitation: for, since I have been able to detect the latter with certainty, I have found the mitral diastolic murmur to be exceedingly rare. It was stated in the first edition of this work that "a slight contraction, such as, for example, to diminish the circumference by a quarter, or from that to half an inch, does not occasion any appreciable murmur with the second sound; for the blood has still sufficient space to pass with tranquillity." It was likewise stated that "a contraction of the mitral or tricuspid valve to the size of only two, three, or four lines in diameter, I have frequently known to occasion little or no murmur" with the

second sound. Much subsequent investigation (in the course of which I have met with very few cases of this murmur) has led me to ascribe its feebleness when it does exist, and its absence in circumstances under which it might have been expected, to the weakness of the current of blood flowing during the diastole from the auricle into the ventricle. This weakness allows the blood to pass in silence through the aperture when only slightly contracted; and when the weakness is preternaturally augmented by debility of the heart, even a high degree of contraction is unproductive of sound. Thus, in Christian Anderson, the tricuspid valve was a thick cartilaginous ring, admitting the middle finger; and the mitral valve, a similar ring, admitting the little finger; yet, as the action of the heart was exceedingly feeble, the pulse and impulse being imperceptible, these lesions were unattended with diastolic murmur, though productive of a loud systolic one from regurgitation. Similar remarks apply to the cases of Sharpe and of Mrs. ——l——n. One of the best marked cases of the murmur in question which has occurred to me for some years, I examined a few days ago. In N. . ., Esq. there was a loud systolic murmur from regurgitation through the mitral valve, followed by a soft, subdued diastolic murmur, louder an inch above the apex than elsewhere, and gradually decreasing on ascending to the sigmoid valves, which did not present any murmur from regurgitation for which the other could be mistaken.

Will it be said that the auricular contraction, previous to the ventricular, should create a murmur? I have looked for it carefully, and have only once been able to suspect it, without being able to assure myself of its existence. Theoretical reasoning seems to countenance this result of observation; for, as the auricular systole is slight (See Exp. p. 18), the quantity of blood injected by it is not considerable; and as the ventricle is already *full*, it cannot admit that extra quantity necessary to bring it to the state of distention, without offering a resistance to its ingress which must greatly retard the force and velocity of the current:—a force, indeed, which can never be great, because the auricles are not only weak muscles, but are unsupported by valves behind.

Tricuspid Valve. 1. Systolic murmur, or from regurgitation. I have seen this occasioned by cartilaginous contraction of the valve to the size of the middle finger (Anderson). Dr. Elliotson mentions two or three cases of adhesion of the valve to the ventricular walls, permitting regurgitation. I have several times seen the same in the dissecting-room. Dilatation of the ventricle, by enlarging the orifice, may create patency of the valves. Systolic murmur of this valve is rare, 1. Because valvular disease does not occur oftener than about once on the right side of the heart for sixteen times on the left; 2. Because, when it does occur, it is almost always much less in degree and usually not sufficient to disable the valves.

The murmur may be loud, because it is occasioned by the considerable power of the right ventricular systole, and because, being nearer the surface, it is more audible than a mitral murmur of equal intensity.

2. Diastolic murmur of the tricuspid valve. This is so rare that (abstracting pulmonic and aortic regurgitant murmurs, with which it is apt to be confounded) I am not satisfied that I have ever met with an instance of it. In Anderson it did not exist, though the aperture was a ring admitting the middle finger. The reason assigned for the rarity of diastolic murmurs in the contracted mitral valve, namely, the feebleness of the current of blood, applies equally to the tricuspid.

Such are the circumstances under which I have noticed valvular murmurs. We now proceed to consider—

The Mechanism and Varieties of Valvular Murmurs.

Valvular murmurs are occasioned by collision of the particles of the blood against each other, and against the containing solids, when this fluid is, by any cause, thrown into preternatural commotion during its passage through the orifice of a cavity. This commotion produces sonorous vibrations in both the fluids and the solids.* To offer an experimental exemplification of this—

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* M. Bouillaud expresses the same idea in the following terms: "The *element* to which we ought, rationally and experimentally, to refer the bellows-murmur that accompanies contraction of an orifice from induration of the valves, is, an increase of friction during the passage of the blood through the orifices or cavities of the heart" (Traité I. 182.). Dr. Corrigan is mistaken in thinking that all explanations of bruit de soufflet have been imperfect because they did not embrace the two following "conditions, which (in his opinion) constitute the mechanism of bruit de soufflet: 1st. *A current-like motion* of the blood (instead of its natural equable movement),

a similar murmur is produced when water is transmitted with sufficient velocity through a tube, in any part of which there exists an internal prominence or contraction of its calibre. The same occurs when the leather pipe of a fire-engine is slightly compressed with a finger; or when similar compression is exercised with the stethoscope on a superficial artery of primary or secondary magnitude, as the subclavian below the clavicle, the carotid, the femoral, &c.; or when the denuded aorta or pulmonary artery is compressed, as in the ass poisoned with woorara (Exp. p. 34, Obs. 14).*

Murmurs present several varieties, which Laennec has desig-

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tending to produce corresponding vibrations in the sides of the cavities or arteries through which it is moving." Now, what is *a current-like motion* but another name for the "preternatural commotion," the "increase of friction" above expressed? Dr. Corrigan proceeds—"2dly, A diminished tension of the parietes of the arteries or cavities themselves, in consequence of which, these parietes are easily thrown into vibrations by the irregular currents of the contained fluid" (Dublin Med. Jour. x. 180). Now, this condition is incorrect. For it has been proved by Mr. Wheatstone that the vibration of solids is not indispensable to the production of murmurs, since he has produced them in the most rigid cast-iron tubes by the vibration of liquids alone. Nor has Dr. Corrigan been fortunate in the selection of the following instance as the strongest foundation for his argument: "In narrowing of the auriculo-ventricular opening of the heart," says he, "the two conditions necessary to generate the sound are in high perfection; and hence, of all the lesions with which bruit de soufflet is connected, it is in this that the sound is most constant" (Ibid. p. 183). I have shown above (p. 75) that this is a reproduction of the old fashioned error of Laennec, resulting from aortic regurgitant murmur being mistaken for direct mitral; and that the latter is, of all murmurs, one of the least frequent and constant. So great a mistake is more surprising in Dr. Corrigan, since he has written on aortic regurgitation as a supposed new disease.

The fact, then, respecting murmurs briefly is, that they may be produced by the vibrations either of the liquid alone, or of the liquid and solids conjointly; and the latter is without doubt the more frequent case in the heart and arteries, because these solids are elastic. Hence it is, that vibratory tremour is in many instances perceptible to the touch.

* It is remarkable that, though Laennec adopted the view that bruit de soufflet of the heart and arteries was referable to "a sort of spasm or tension," the true view did not escape him; for he mentions as the other alternative which he rejected, that the murmur "owed its origin to a particular state of the blood, or *to the manner in which this liquid was moved*" (Traité, ii. 429). Dr. C. J. B. Williams is therefore mistaken in supposing himself to have been the author of the conjecture that bruit de soufflet was referable to "the motion of liquids in, or against solids of a particular form" (on the Pathol. and Diag. of Diseases of the Chest, p. 193. 1835). He is also mistaken in supposing that I adopted his view; for, as explained in a note at p. 58 of this work in 1831, my own opinions were in print before I had seen the above words in his Rational Expos. p. 50.

nated by the epithets *bellows-murmur*, *sawing or filing*, *rasping*, *a continuous murmur like that in a large sea-shell*, and *a whistling or musical murmur*. By bellows-murmur, he meant a smooth, soft tone, like that of blowing with hand-bellows. By sawing, filing, and rasping, he meant to indicate merely successive degrees of *roughness* (Traité, ii. 423), without including, as is too commonly imagined, the pitch or key of the note. M. Bouillaud does not seem clearly to have understood this distinction: at least, he has not clearly marked it (Traité, tom. i. 167 and 187). Thus, he says that whispering the letter *s*, is an exact imitation of the sawing-murmur; but that a similar whisper of the letter *r*, is thicker (plus *gras*) than the sawing sound. Here, he evidently considers the key, and not the roughness, to be the characteristic of the sawing sound. A slight degree of roughness, Laennec designated by comparing it to the distant filing or sawing of wood; and a high degree, by comparing it to the rasping of wood.

Laennec imagined that the rougher murmurs of filing, sawing, and rasping denoted ossification; and that the softer or bellows-murmur was connected with obstructions presenting a smooth surface as the fibrous and cartilaginous—a view which MM. Bertin and Bouillaud adopted, and which M. Bouillaud does not wholly discard in his later work (Traité, i. 187). I feel confident, however, from the examination of a great number of cases, that the view is not so correct as to admit of being adopted as a general rule. It is true that rough, denuded, salient ossifications will occasion a rasping murmur, because they effectually break the current of the blood; but I have repeatedly known less prominent ossifications, especially when still covered by the lining membrane of the heart, occasion merely a soft bellows-murmur. On the other hand, in innumerable cases, I have found sawing, filing, and even rasping murmurs produced by merely fibrous or fibro-cartilaginous disease, which, in many cases, I ascertained by dissection, but, in others, merely inferred from the youth of the patient or the recent date of the disease. Dr. Elliotson imagines that *degree* of contraction is the sole essential to roughness (Lum. Lec. p. 15). That a considerable degree of contraction is an important essential, I do not doubt; but that the roughness is always in the direct ratio of the degree of contraction, is a proposition from which I am bound to dissent; for I have habitually found the highest possible degrees of contraction attended with soft bellows-murmur. For instance, mitral regurgitations, often occurring through a chink so small as not to impair the strength of the pulse, frequently yield a perfectly soft, though possibly a loud bellows-murmur, for softness and loudness are by no means incompatible circumstances.

Hence I conclude that the roughness of murmurs is neither connected with any particular anatomical element of the valvular lesion, nor directly proportionate to the degree of contraction; but that it depends upon such an accidental *configuration* of the contracted orifice as is best calculated to break the stream of the blood, and throw it and the contiguous solids into large vibrations—a configuration with which I have generally found a considerable, though not necessarily a high degree of contraction to coincide.* Considerable strength of the current, however, is a further essential to roughness; as is shown by the facts that no roughness attends the murmurs from sigmoid regurgitation or influx from the auricles into the ventricles, and that rasping murmurs may often be made temporarily soft by bleeding, digitalis, &c.

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* How much the discharge of fluids is connected with the configuration of the apertures, is illustrated by the experiments of Venturi. He found that any vessel or reservoir discharged less through a simple, circular hole in its base—viz. only 62 quarts in 100 seconds, than through one to which was affixed a short tube, of the same diameter as the hole, and twice the length of its diameter, from which the discharge was 82 quarts. He found, again, that, if the tube was pushed up some distance into the vessel, the flow of water was diminished, even to less than issued from the simple aperture, namely, to less than 62 quarts.

Sir Isaac Newton had previously ascertained, that fluid, tending from all parts of a reservoir to one common centre or orifice in the bottom, proceeded in curves. It occurred to Venturi that, such being the natural form in which water tends to discharge itself, a pipe of that form would favour the discharge: and, accordingly, he found this to be the case, the amount being 98 quarts. Conceiving, further, that the curve in which water naturally tends to an orifice, was, from the inertia of water, continued beyond the point of discharge, he made the pipe trumpet-mouthed beyond its narrowest point, in the same curve as before it; and from this he obtained the maximum discharge.

These differences in the quantity of discharge from orifices of the same area, depend on the degree in which the currents cross each other at the orifice, and thus constitute a greater or less obstruction to the direct passage of the whole body of fluid. It is obvious that the degree of obstruction will be greater in proportion as the currents cross at greater angles, and that it will be still further increased by any counter currents or eddies opposing the converging currents. It will be easy for the reader to see the application of these experiments to the various valvular contractions.

To sum up, then, the presumptions afforded by a rough or rasping murmur would be 1. that the cause is organic, for inorganic murmurs, as will hereafter be shown, are never rough; 2. that the contraction of the orifice is not inconsiderable; 3. that if the murmur occur after the age of 60, the disease is probably osseous.

The continuous murmur. Laennec says, "In very rare cases, the bellows-murmur changes, in the carotids especially, and even in the heart, into a continuous murmur analogous to that of the sea, or to that which we hear on placing near the ear a large univalve sea-shell" (Traité, ii. p. 422). I shall hereafter prove that Laennec and his follower Bouillaud have been mistaken in referring continuous murmurs to the arteries, their real seat being in the veins, where, so far from being rare, they are very common. Laennec does not allude to the circumstances under which continuous murmur occurs in the heart, nor have they, to my knowledge, been explained by other authors: I shall therefore offer the results of my own observation. I have twice heard continuous murmur in the heart, in a very marked degree. In one case (Jones), it depended on a moderate quantity of fluid churned, as it were, in the pericardium rough with lymph. In the other case (Mitchell), it was occasioned by regurgitation out of an aortic aneurism into the right ventricle. In both, though continuous, it was augmented in intensity during the ventricular systole and diastole; and in both it was attended with a great degree of purring tremour.

I have two or three times met with continuous murmurs, augmented synchronously with the pulse, along the tract of the pulmonary artery, apparently in connexion with dilatation of the vessel (case of Miss L. P.); but I suspect that while the augmentations were seated in the artery, the continuous portion of the murmur resided in the vena innominata, compressed or displaced by the dilatation. This subject will be explained under nervous murmurs. See especially the case of James.

The bellows, sawing, rasping, and continuous murmurs in the heart are louder, cæteris paribus, in proportion as the stream of blood through the contracted orifice is stronger. This, which is obvious on theoretical grounds, I have found amply confirmed by observation. Thus, murmurs are increased by accelerating the heart's action, and diminished by calming it, especially if the pulse be much lowered by digitalis. Again, I have collected six or seven cases of valvular disease, in which there was one strong contraction of the ventricles producing a pulse, followed by two or three feeble contractions attended with a barely perceptible pulse. The strong contractions occasioned a murmur; the weak, none. Again, the currents by regurgitation through the sigmoid valves, and those flowing out of the auricles into the ventricles through the contracted auricular valves, are more feeble than the currents setting in the opposite directions, that is, out of the ventricles; and the corresponding murmurs I have invariably found to be weaker. The strength of the current, however, is not the only circumstance which occasions loudness of murmurs; for such a configuration of the stricture as most breaks the stream, produces not only a rougher murmur, as already shown, but one of greater intensity. Accordingly, we find rough murmurs, cæteris paribus, louder than others, with the exception of musical tones, these being, from their acute nature, more calculated for transmission to a distance.

The pitch or key of the bellows, filing, sawing and rasping murmurs (as distinguished from their roughness) depends mainly on the depth or distance from the surface at which murmurs are generated, the pitch being higher in proportion as they are nearer, and *vice versâ*; but it is also slightly elevated by a stronger current and depressed by a weaker. A very narrow aperture also raises the key, provided the current be strong. These circumstances were not pointed out by Laennec or the other French writers; whence has resulted the prevailing confusion respecting the meaning of the above epithets, filing, sawing, &c. After much attention devoted to this point, I think that the following characters will be found at once tolerably accurate and easy of comprehension.

Murmurs seated in the pulmonary orifice or artery, from being the most superficial, are on a higher key than any others. Though they are seldom so high as the whispered letter *s*, yet they range between this and the whispered letter *r*. Murmurs originating in the ascending aorta where it approaches near to the sternum, are for the same reason on almost as high a key.

Murmurs in the aortic orifice, being rather more deeply seated, seldom rise higher than a whispered *r*, which is their average key, and it is perhaps the most ordinary type of the sawing sound. M. Bouillaud, however, (to whom I am indebted for the ingenious idea of representing sounds by letters and who has used *s* and *r* for this purpose) thinks that *s* more truly represents the sawing sound.

Murmurs from aortic and pulmonic regurgitations, in consequence of the currents being weaker, are generally two tones lower, like whispering *awe* by inspiration, and the click of the valves, when audible, may be represented by prefixing the letter *p*, as in the word *paw*.

Murmurs in the mitral valve, from being still more deeply seated, are on the average four tones lower, like a whispered *who*: the tone is somewhat elevated by a very strong current, as that of violent mitral regurgitation, and depressed by a feeble current, as that producing diastolic mitral murmurs.

Tricuspid murmurs are rather higher toned than mitral, because less deeply seated.

It is scarcely necessary to explain that the depth or "hollowness" of murmurs is referable to remoteness and reverberation through the chest. On this principle we should anticipate, and observation proves, that a murmur is low-toned, not only from being deeply seated, but also from being explored at a distance. Thus, an *r* toned murmur generated in the semilunar valves, sounds as low and remote as a whispered *who* if explored above the clavicles, an inch on either side of the sternum or near the apex of the heart.

The principal use of this knowledge of the pitch or key of murmurs, is, to enable the auscultator to trace a murmur up to its source—the point where it sounds loudest and seems *nearest* to his ear; for, without this ability, he can never succeed at particular valvular diagnosis. Though the rules offered have required many words for their development, they are, practically, so simple, that a student, if well taught on three or four marked cases, can make himself master of them in half an hour.

Musical murmurs. This variety, says Laennec, occurs in the arteries only, or at least I have never met with it in the heart." This acute observer is mistaken in both these propositions. I shall hereafter show (see *Venous Murmurs*) that the musical murmur which he, M. Bouillaud, and all other writers have ascribed to the arteries, is really seated in the veins and is a twin phenomenon with the continuous murmur. It is my present object to show that the musical murmur is also a common occurrence in the heart, that it may be a perfect note like whistling, cooing, or the mewing of a kitten, and that it frequently co-exists or is blended with an ordinary murmur. In the first edition of this work, I described the case of a patient who applied to me for "a noise in the chest," which I found to be a musical note, audible at the distance of no less than two feet. I added that, in a precisely similar case which I found described by Dr. Elliotson in the Med. Gaz. of the week in which I was writing, a very large and long vegetation existed in the mitral valve. As I was writing in 1830, before the publication of Dr. Elliotson's Lum. Lectures, I imagined that these were the two first recorded cases of musical murmur in the heart; but, on reading his Lectures some years afterwards, I found reason to believe that he had observed the phenomenon at an earlier period than myself; and I take the present opportunity of making this acknowledgment, as I was not aware of the fact when the first edition of the present work issued from the press. "I have," says Dr. Elliotson, "heard it (the musical murmur) exactly resembling the cooing of a dove—a variety not mentioned, I believe, by authors. In one case it was so loud that I heard it when standing nearly a foot from the patient. Three times have I heard this cooing sound." He subjoins in a note, "A fourth instance has lately fallen under my notice." This was, I doubt not, the one which I found in the Med. Gaz.

Since I first heard the musical murmur in 1830, I have met with fourteen or fifteen instances of it. In some it was an almost pure note; but in the majority it was blended with more or less of an ordinary bellows-murmur: and in some of the latter the musical note seems to have been coeval with the concomitant murmur, while in others it commenced later, and in others again it ceased, and was replaced by a murmur. Four cases are appended to illustrate these several varieties. Thus, in the interesting case of Milton, the musical note was a clear tone like the *oo* in *coo*, swelling and also rising a semitone in the middle, like the mew of a kitten. It attended the second sound and proceeded from aortic regurgitation. A feeble sighing murmur, occasioned by the refluent stream within the ventricle, was heard down the tract of the ventricle, (but not above the valves,) accompanying and prolonging the musical note. In the case of V. Esq., there was a mixed musical and ordinary murmur. In the case of Jones, the musical note was a broken whistle, very loud and distinct, and it ultimately degenerated into a loud sawing murmur. Its cause was mitral regurgitation. In Tindal, on the contrary, an ordinary murmur took precedence, and the musical note supervened at a later period. From these cases it is clear that ordinary and musical murmurs are identical phenomena resolvable into each other, the latter merely consisting of finer and more even vibrations. To use an illustration which I have employed for years (and which Bouillaud, who takes the same view, has also employed) there is the same and no other difference between the two sounds than between a blow and a whistle, as performed by the lips; and no artifice is easier than that by which we make the transition from the one to the other. That the medium is liquid in one case and aeriform in the other, is unimportant; for M. Lagniard Latour has succeeded in producing musical notes by the flow of liquids through apertures in tubes.

A musical murmur, therefore, indicates nothing more than an ordinary one. I am inclined to think that it is most apt to result from regurgitation. All my own cases, except two or three, have been of this nature; and the presentation of an edge to a stream, is best calculated, according to Mr. Wheatstone, to produce musical vibrations. Dr. Elliotson states that in all his cases, the cooing accompanied the ventricular diastole, and was referable in situation to the mitral valve; but as he was then unacquainted with the regurgitations, it is more than probable that he mistook the murmurs of aortic regurgitation for mitral diastolic murmurs,—for I have shown that murmurs from the latter cause are extremely rare. M. Bouillaud (who is mistaken in supposing that "the musical whistle of the heart had not yet (in 1835) been noticed by any one to his knowledge") gives seven cases of this phenomenon, in one of which it proceeded from mitral regurgitation, in another it accompanied the first sound in the aortic orifice, and respecting the remaining five, he is silent (Traité, i. 168. His first case was in 1828).

From all that has now been advanced respecting valvular murmurs, a subject on which I have dwelt with the desire to simplify it by the introduction of fixed general rules, the following conclusions may be deduced.

1. The ventricular *systolic* currents through contracted orifices, from being stronger than the *diastolic*, produce louder murmurs.

2. Considerable contractions, of a rough, salient configuration, whether osseous or not, produce the rough murmurs of sawing, filing, or rasping, provided the current be that of the ventricular systole, its diastolic currents being too feeble.

3. The pitch or key of murmurs is higher in proportion as they are generated nearer the surface, and the currents producing them are stronger; and *vice versâ*. Also, the key is lowered by distance, independent of depth, from reverberation through the chest.

4. Musical murmurs indicate nothing more than ordinary murmurs.

5. Rough murmurs, and even loud and permanent bellows-murmurs, indicate organic disease.

6. Murmurs from regurgitation necessarily indicate organic lesions.

7. Continuous murmurs in the heart will probably be found to indicate, sometimes organic disease attended with regurgitation out of the aorta into the right ventricle or pulmonary artery; sometimes churning of a little serum between layers of rough lymph on the pericardium; and sometimes, probably, dilatation of the pulmonary artery and compression of the vena innominata.

It has been explained above that a knowledge of the key or pitch of murmurs assists the auscultator in tracing them up to their sources, or, in other words, up to the situations in which they are most audible. It remains to be explained what these situations are in reference to the several valves, and this constitutes beyond comparison the most important essential to particular valvular diagnosis.

Situation in which Murmurs of the respective Valves are most audible.

Authors had not pointed out these situations with any degree of accuracy previous to the first edition of this work: nor was it possible for them to do so; for, as they were unacquainted with the regurgitations, they could not know whether a murmur with either sound was seated in an arterial, or in an auricular orifice. Dr. Elliotson, indeed, who had heard of the regurgitations, but had no *practical* acquaintance with them, attempted, in his Lum. Lectures in 1830, to define the situations in question; but the subjoined quotation will at once evince his total failure, and the necessity for more precise rules on the subject.* In the appendix to the second edition of this work, I made corrections and additions to my previous rules, being greatly assisted by the strong light reflected on the subject by my experiments demonstrating the causes of the natural sounds. I am now enabled to offer a code of rules of so simple and obvious a nature that, with the assistance of the diagram Fig. 4, I have found students acquire them in the course of a few minutes.

Murmurs seated in the semilunar valves are best heard immediately over those valves, (that is, on the sternum, opposite to the inferior margin of the third ribs when the patient is horizontal, and a *little* lower when he is erect), and thence for about two inches upwards, along the diverging courses of the aorta and pulmonary artery respectively. A distinct murmur high up the aorta proceeds from the aortic valves, as a pulmonic murmur is only feebly and indistinctly transmitted in that direction. It may be known that the murmur proceeds from the aortic valves rather than from the diseased ascending aorta itself, by its key not being higher than a whispered *r*, whereas a murmur from the aorta itself is commonly a tone or two higher, approaching towards an *s*, and also seems much *nearer* and more superficial.

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* "If the impediment is in the left ventricle, at the mouth of the aorta, it" (the murmur) "is *loudest at the cartilages of the ribs* to the left of the sternum; if in the right ventricle, at the mouth of the pulmonary artery, it is loudest at the sternum and to the right. The sound is often so loud, that it prevents the natural sound of the auricles from being distinctly perceptible till the ear or stethoscope is removed from the region of the ventricles, higher, to the region of the auricles."

"When the impediment is at either of the auriculo-ventricular openings, the morbid sound is heard at the moment of the auricular contraction" (*i. e.* with the second sound), "and is generally loudest at the *superior part of the cardiac region*. It is loudest at the cartilages of the left ribs, when the left auriculo-ventricular opening is narrowed; loudest at the sternum and to the right, when the narrowing is at the right auriculo-ventricular opening." Nothing can be more erroneous, contradictory, and confused than this account. The subject was, in fact, necessarily inexplicable till the regurgitations and immediate sources of the murmurs became known.

A distinct murmur high up the pulmonary artery proceeds from the pulmonic valves, as an aortic murmur is only feebly and indistinctly transmitted in that direction. The pulmonic murmur, whether seated in the valves or in the pulmonary artery itself, (as when dilated,) always sounds near and superficial, provided the current be sufficiently strong; because the valves and artery are close to the surface, the valves being not only in front of the aortic valves, but half an inch higher up. A murmur in the pulmonic orifice is more audible down the tract of the right ventricle than of the left—which is a corroborative circumstance.

Thus, by listening *high up* the aorta and pulmonary artery, it is easily ascertained in which vessel the murmur is seated. This rule will even apply to semilunar regurgitations, notwithstanding that their murmurs are weaker and not so well transmitted *up* the vessels in consequence of the current setting *out* of them into the ventricles. Further rules for distinguishing these regurgitations have been offered at p. 74. There is a further and most important advantage in exploring murmurs of the semilunar valves high up the vessels: namely, that in these situations murmurs of the auricular valves are, from their remoteness, either wholly inaudible or very obscure: although, therefore, an auricular murmur should co-exist, it would not prevent the auscultator from deciding that a loud and near sounding murmur, heard high up the vessels, was generated in or above the arterial orifices.

Murmurs seated in the auricular valves are best heard at that part of the præcordial region where, from the heart being in contact with the walls of the chest, there is dulness on percussion —in short, about the apex; for the murmur is best conducted to the surface through a solid medium. The upper and *left* side of the dull portion, being nearest to the mitral valve, is the best point for exploring its murmurs; and this point will generally be found situated about the fifth rib or subjacent intercostal space, and a little to the right of the nipple: in females, it is under the mamma when pretty well raised, and a little to the right of its centre. If the impulse of the heart be perceptible, there is no better guide than this to the situation in question. The auscultator has only to place his stethoscope about an inch above the spot where the apex impinges.

The upper and *right* side of the dull portion, being nearest to the tricuspid valve, is the best point for exploring the murmurs of this valve; and the point will generally be found on or near the sternum, at the same level as on the opposite side. If, in making these explorations of either valve, the stethoscope be placed half over the dull portion and half over the thin resonant edge of the lung, the object will be sufficiently answered.

There is a further, and most important advantage in exploring murmurs of the auricular orifices in these low situations: namely, that the murmurs sound so *near* and distinct as to preclude the idea of their being generated in the arterial orifices, the murmurs of which always sound *remote* and obscure when explored near the apex of the heart. The only source of fallacy is in the case of regurgitation through the semilunar valves on either side of the heart; for, here, the murmur descends down the ventricle with the refluent stream. It has been shown that this was the fallacy which deceived Laennec, Bouillaud, Elliotson, and all others who have believed in the frequency of diastolic murmurs of the auricular valves; yet it is obviated with the utmost ease by attention to the fact that the regurgitant murmur increases progressively on ascending from near the apex to the semilunar valves, and that it is audible above them; whereas, the auricular diastolic murmur decreases in the same progression and is totally inaudible above them.

When both the semilunar and the auricular valves are diseased, it is perfectly easy to ascertain this by observing, according to the above rules together with those for the pitch of murmurs, that there are two distinct sources of murmur.

When two murmurs are seated in the same orifice, this is readily ascertained by tracing them up to the single source, and noticing that one attends the first, and the other the second sound.

In making a valvular diagnosis, it is necessary to keep the finger constantly on the pulse, in order to distinguish the first and second sounds, with their murmurs, from each other. If the radial pulse be much later than the first sound, the carotid should be felt, as its synchronism is more perfect. It is necessary to reiterate these obvious rules, because, from inadvertence, they are habitually neglected by novices.

In exploring a delicate murmur, the auscultator should hold his own breath and request the patient to do the same. The utmost attainable silence should reign in the room. If an expert auscultator can hear in a noise, it is because he catches the sound during the momentary intervals of silence, but the learner must not expect to accomplish this. He should always endeavour to keep his head erect and his neck straight, otherwise cerebral congestion will impair the nicety of his hearing. Many vaunt the superiority of the naked ear over the stethoscope. The writer has not found himself inferior to others in the use of the naked ear, but he may perhaps be permitted to say that he possesses far more delicacy with the cylinder than without it; whence he suspects that those who entertain an opposite opinion, unconsciously labour under some special disadvantage. He has observed the disadvantages to be principally of three kinds: 1. The inexpert scarcely ever apply the instrument air-tightly; 2. the stethoscope is a bad one—the ear-piece nearly flat, the joints loose, the cone false and the bore unpolished;* 3. the auscultator is dull of hearing. An eminent practitioner and even a teacher of auscultation with respect to the lungs, remarked to me that he did not believe that anything had yet been done to unravel the murmurs of the heart. I felt surprised. My surprise ceased when he subjoined that "for his own part he had never yet been able to distinguish the two natural sounds of the heart!"

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* Finding so many bad instruments in use, in 1833 I taught a clever turner (Grumbridge, 42, Poland-street, Oxford-street) to make stethoscopes, and he now makes a greater number, and incomparably better instruments than perhaps any one in the metropolis, or the kingdom, —completely avoiding all the defects specified in the text. The two kinds I recommend, and for which he has my models, are, the long, thick one without a joint, for home and hospital practice (price 4*s.*); and the long, thin one with a screw-joint in the middle, for carrying in the pocket (price 7*s.* 6*d.*) I would dissuade the student from accustoming himself to a short stethoscope; first, because it possesses little if any advantage over the long one; secondly, because in private practice a long one is more agreeable to both parties on the score of delicacy; and thirdly, because a short one is exceedingly inconvenient in reaching over large beds, &c. and is often the cause of a slovenly exploration. The only innovation which I have ventured to make in the construction of the stethoscope, consists in a deeper excavation, larger circumference, and a more bevelled or rounded edge of the ear-piece, than was employed by Laennec. Such an ear-piece suits almost every one on the first trial. Its advantage consists in its being air-tight when applied, and in its bringing a large surface of the ear into solid contact with the cranium; for, as Dr. Cowan has ably shown, the solids as well as the meatus externus conduct the sound.

SECTION III.

MURMUR FROM HYPERTROPHY WITH DILATATION, AND ITS MECHANISM.

In a case which presented itself to me in 1825 (Med. Gaz., Sept. 5, 1829, p. 420), I was led to notice that murmur was produced by a disproportion between the cavities and the orifices, consequent on enlargement of the former. Laennec also mentions bellows-murmur from hypertrophy or dilatation (Traité, ii. p. 441, second edition). In the former editions, I represented it to be of frequent occurrence in the aortic orifice and with the first sound, in cases of great hypertrophy with dilatation; but I have subsequently found that it is restricted to those cases almost exclusively in which there is anæmia, a state very apt to supervene in the advanced stages of organic disease of the heart, and which will be shown in the ensuing section to be the principal cause of murmurs independent of organic impediments. The changed form of the ventricle in hypertrophy with dilatation probably co-operates in the production of the murmur; for, as the cavity is more spherical than natural, and its artery consequently rises more abruptly with respect to its internal surface, the currents of blood reflected from its sides meet in the orifice at more obtuse angles, and thus, by their collision, not only give rise to the murmur, but impede each other's passage into the vessel. For the latter reason, the pulse is sometimes small and weak, when the impulse of the heart is violent,—a paradox with which authors have been much perplexed.

SECTION IV.

MURMURS IN THE HEART AND ARTERIES INDEPENDENT OF ORGANIC DISEASE.

Before proceeding to assign the cause of this phenomenon, it is necessary to be agreed as to the circumstances under which it occurs. The account which Laennec gives of it, and of the concomitant phenomena purring tremour *(frémissement cataire)*, and (what I conceive to be merely a less degree of the same) thrilling (frémissement) of the arteries, does not accord with my own observation, and it involves several inconsistencies, which render the phenomena equally inexplicable on his own and on every other theory. To question anything which Laennec explicitly states as a fact, is hazardous: the more I have studied his works, the more have I become sensible of this, and felt astonished at the wonderful accuracy of his powers of observation. With respect to the subject before us, however, it is both apparent from the statements in his treatise, and well known to those who were acquainted with him, that he had not satisfied his own mind: that he was conscious of incongruities which he could not reconcile, and of difficulties which he was unable to surmount. With less presumption, therefore, may I enter on an investigation which his genius can only be said to have left incomplete; and I do it with more satisfaction, as I have to advocate the cause of auscultation against its great inventor, and to show that the doctrines broached in his first edition respecting bellows-murmur as a sign of valvular disease, were not, as he imagined, invalidated by the more extended knowledge of the nature of this phenomenon which he supposed himself to have acquired at a later period.

" The bellows- murmur," says Laennec, " may accompany the *diastole* of the heart and that of the arteries, and it is connected with them in such a manner as to replace and entirely annihilate their natural sound (*i. e.* the second); so that, at each diastole, the ventricle, the auricle, or the artery in which the phenomenon takes place, yields a distinct sound of a puff of the bellows, the noise of which ceases during the systole" (De l'Auscult. t. ii. p. 422).

This account is clearly inconsistent with itself. It is certain that the murmur in question takes place synchronously in the heart and arteries: it cannot, therefore, take place during the *diastole* of both, as the diastole of the one coincides with the systole of the other. The error consists, as I shall presently show, in saying that the murmur coincides with the diastole of the ventricles, instead of with their systole. Granting, for a moment, that the murmur does, as Laennec imagines, accompany the diastolic movements of the ventricles, this view is irreconcileable with his explanation of the cause of the phenomena; for, having disavowed his belief that the cause is connected with the motions of the fluid (De l'Auscult. tom. ii. p. 429), he says, "The perfect similitude of the intermittent *muscular sound* (bruit musculaire) and of the bellows-murmur of the heart and arteries, appears to me entirely to decide the questions which I have above proposed on the nature of this murmur, and to prove that it is referable to a real spasmodic *contraction*, whether of the heart or of the arteries. The possibility of a spasm of the heart needs not be demonstrated, since that organ is muscular. With respect to the arteries, the circular fibres which compose their middle coat appear to announce a tissue endued with the faculty of contraction." (Ibid. p. 440.) Now, if spasmodic *contraction* be the cause of the bellows-murmur, this murmur cannot take place during the diastole of the heart, which, according to the best authorities, is an act, not of contraction, but of relaxation. Neither will spasmodic contraction account for the bellows-murmur in the arteries; for the murmur takes place during their diastole, and not during their systole, as Laennec's theory supposes. Apparently conscious of this inconsistency, he endeavours to reconcile it by saying that the murmur occurs while the artery is in the act of turning from its diastolic to its systolic state. There is, however, no doubt that it occurs while the artery is in the progress of dilatation.

The cause of the confusion and inconsistency into which Laennec has fallen, is evidently that to which I have so often adverted—his unacquaintance with the regurgitations. Thus, the quotation from this author at p. 95, is manifestly a description of semilunar regurgitation. That he should have overlooked this regurgitation, is not surprising, for two reasons: 1. because the organic lesion of the valves producing it is often slight, —nay, sometimes totally absent; for the regurgitation may result from mere dilatation of the orifice: 2. because his attention in the post-mortem examination was wholly directed to the auriculo-ventricular valves, disease of which he believed to be the only organic source of murmurs with the second sound. Detecting no organic disease in these valves, it is not wonderful that he should ascribe the murmur to inorganic causes: nor is it wonderful that the frequency of these supposed inorganic murmurs should have made a strong impression on his mind; for I have already shown that semilunar regurgitation is one of the most common forms of valvular disease. In studying genuine inorganic murmurs, therefore, we must carefully exclude this source of fallacy.

Respecting the purring tremour (frémissement cataire) of arteries, Laennec avows that, notwithstanding all the pains he has taken for the purpose, he has not been able to discover any satisfactory reason for the phenomenon (De l'Auscult. tom. ii. p. 452). Nor is this surprising: for having attributed the twin and concomitant phenomenon, bellows-murmur, to spasm,—a state tending to place an artery in a state of constriction and immobility, rather than of vibration, he has no other physical resource remaining, by which to explain the purring tremour. He accordingly yields to the difficulty; for it is little more than a substitution of words for ideas, to say, "it is at least extremely probable that the purring tremour depends upon a particular modification of the nervous action" (innervation) (ibid. p. 453); and that "the three phenomena, bellows-murmur, purring tremour, and the thrilling pulse, are attributable to different, though analogous, modifications of the action of the arteries and the heart, and that the one cannot be regarded as a more or less intense degree of the other" (ii. p. 767). M. Bouillaud has more recently made similar strictures on Laennec. "Having," says he (Traité, i. 229, 1835), "made vain efforts to untie this sort of Gordian knot, like another Alexander he has cut it. In twenty places of his work, we see him repeat the same profession of faith: there, he says that the different varieties of arterial bellows-murmur are due *to a peculiar vital state of the arteries* (ii. p. 429); here, that they are *due to a spasm of the arteries* (p. 441—443); elsewhere, that they depend *on a simple modification of the innervation—an anomaly of the nervous influx*" (p. 763).

Having thus endeavoured to present a brief sketch of a subject, which, from its obscurity, has in general occupied several chapters, I proceed to offer an explanation of the inorganic murmurs, tremours, and thrills in question on a different principle; and I trust to show that it is possible to surmount the physical difficulties of which even M. Bouillaud complains, and which have prevented him, no less than Laennec, from bringing the subject to a satisfactory conclusion.

As my own experience does not accord with that of Laennec as to which motions of the heart are accompanied by the murmur, it is necessary to premise that I have found it accompany the *systole* of the ventricles exclusively. In the arteries, it coincides with their diastole, which is synchronous with the ventricular systole. The purring tremour occurs at the same moment and is a result of the same cause. The arterial thrill is nothing more than a less degree of the purring tremour.

Both by experimental and pathological evidence, I am led to believe that the murmurs and tremours, as well in the heart as in the arteries, are occasioned by modifications in the motion of the fluid, occasioning increased friction and vibration. To establish this point, it is necessary to prove, 1. that liquids permeating tubes, do occasion murmurs and tremours: 2. that, in the living subject, modifications in the motion of the blood calculated to elicit murmurs and tremours, do take place under the circumstances in which such murmurs and tremours actually occur: 3. that the explanation applies equally, whatever be the circumstances under which the murmurs and tremours occur.

1. That a bellows sound is produced by the transmission of a fluid, without any intermixture of air, through a tube, though questioned by Laennec (tom. ii. p. 763-4, note), is a fact too easy of demonstration to require discussion. Having just returned from a repetition of the experiment,—one which I have frequently performed, I find the rushing murmur so distinct and

close to the ear, as to preclude the idea of a fallacy from the movement of a piston or any other cause: I find the sound to vary in intensity according to the velocity with which the fluid is propelled, to be increased by bending the tube at an angle, and to be still further increased, but also modified, by the admission of air—becoming of a rattling nature, totally different from any sound heard in the heart or arteries. A thrill or vibration, perceptible to the hand, attends the murmur provided the motion of the fluid be sufficiently rapid, or provided, with a less rapid current, the interior of the tube be rough or obstructed. The vibration is best felt in thin metallic tubes (from the superior vibratory power of metals), and in yielding tubes, like the leather pipe of a steam engine; and the thrill in the latter is increased by locally compressing or indenting it. These experiments have been performed with the same results by M. Pelletan, professor of medical physics to the Faculty of Paris; by Dr. Spittal on leaden pipes (*Med. Gaz.* Aug. 3, 1833); by M. Piorry (*Percuss. Méd. et Archiv. Génér. de Med.*); and by M. M. Bouillaud and Donné (Traité du Cœur par Bouillaud, i. 205). The three latter gentlemen have even produced the murmur by injecting water into the arteries of the dead subject, and have thus corrected an error into which M. Pelletan had fallen; namely, that vessels with a smooth interior did not yield the murmur.

What experiment thus proves, the principles of hydraulics would lead us to anticipate. It is a fact, established by the investigations of Newton, De Buat, Bernoulli d'Alembert, Robison, Venturi, Dr. Young, and others, that the progress of fluids through pipes, however smooth, is retarded by friction against their interior; and that the retardation is increased by all projections, irregularities, and sudden bends; for the fluid, striking against these, forms reverberations and eddies, which impede its current as effectually as solid obstacles. The friction increases with the increase of velocity, and, beyond a certain point, it increases in a much more than simple ratio. Thus, if a steam engine of ten horse power will propel a vessel ten miles an hour, one of a hundred would not suffice to propel it twenty. Now the friction,—in other words, the collision of the particles of fluid against the sides of the vessel and against each other, by producing vibrations of a certain rate of rapidity, is the cause of the sound; and these two phenomena, the friction and the murmur, are, consequently, in the direct ratio of each other. Hence it appears that the murmur is produced in strict conformity with the general axiom, that the particles of all bodies, when thrown into sufficient vibration, generate sound. I have already shown (p. 80), that Dr. Corrigan's supposed new theory of murmurs is partly incorrect, and that, where correct, it is identical with my own, which he has misunderstood and imperfectly represented (Dub. Jour. vol. x. p. 177). His *current-like motion* is nothing more than the "reverberations and eddies," the "preternatural commotion," the "arterial vibration," described above and in other parts of this work, as attending the production of murmurs. These phenomena, together with the lax arterial coats, the coats of unfilled arteries, have never been more strikingly exemplified than by the experiments detailed under the ensuing head.

2. It is next to be proved that, when murmurs in the heart and arteries do occur independent of organic disease, there is an increase of friction, dependent on a modification of the motion of the blood, to account for them.

Being engaged with Dr. Marshal Hall in a series of experiments on the effects of loss of blood, &c.—a subject for the elucidation of which the profession is much indebted to that gentleman, we took the opportunity of studying the stethoscopic phenomena of the circulation under all the circumstances of collapse, reaction, &c.

Eight or ten dogs were blooded more or less frequently, from once to ten or twelve times, and at intervals varying from twenty-four to seventy-two hours. The results were, that, on the day following the first or second abstraction of blood to the amount of eight or ten ounces, the systolic sound of the heart, previously loud and clear, became attended with a whizzing or sawing murmur, the impulse increased and became unusually smart or abrupt, and the pulse became quick and jerking (the pulse of unfilled arteries), with a thrill and a throbbing, perceptible over the whole body. These phenomena increased up to the fourth or fifth bleeding, when they appeared to attain their maximum, the sawing sound being extremely loud, the impulse and pulse violently jerking and bounding, the arterial thrill or purring tremour excessive. and the throbbing perceptible not only when the finger was placed on an artery, but when the hand grasped a large surface of the body. A hissing bellows-murmur was, moreover, distinctly heard, when the stethoscope was placed over any considerable artery, as the femoral or carotid. The pulse at this time generally beat from 150 to 190 per minute, its natural standard being about 120.

The phenomena underwent the following changes in correspondence with changes in the circumstances. The animals being *extremely nervous and irritable*, the pulse was instantly accelerated ten or fifteen beats per minute by the slightest excitement, as that of being moved or startled; and the murmur and jerk sustained, in consequence, a remarkable increase.

After reiterated venesections the pulse became small and weak; but, so long as it remained jerking, the murmur continued, though not so loud as previously.

If venesection was omitted for three or four days, reaction subsided; and in proportion as the pulse and impulse became softer, though without a loss of *real* strength and fulness, the murmur, both of the heart and arteries, the purring tremour, the general throbbing, and the nervous irritability, gradually disappeared.

If, during the full prevalence of all the phenomena, the animal was bled to the approach of syncope, the pulse and beats of the heart, reduced to about 100 per minute, became feeble and soft, and at the same time lost all murmur and thrill; but, in the course of from fifteen to thirty minutes, reaction was re-established, and all the symptoms recurred.

If the animal was held erect by the fore legs, a posture which, either by diminishing the afflux of blood to the brain, or by obstructing the circulation through the heart and lungs, caused the gradual supervention of syncope, the pulse became slow, soft and feeble, and the murmur and thrill were suspended; but they were promptly restored to their former state when the animal was placed on its legs.

From these experiments it may be concluded, *a.* That diminution and attenuation of the blood are circumstances eminently favourable to the production of inorganic murmurs:—the diminution, because, as the weight of the blood is diminished, the fluid can more easily be propelled with velocity, and because the diminished tension of the arteries allows more latitude for sonorous vibration both of their walls and of the blood: the attenuation, because the particles, having lost a proportion of their lubricity, are better calculated for rapid motion, and consequently for the production of murmur and vibration by collision against each other and against the walls of the containing vessel. This attenuation (the existence of which was strikingly displayed in some of the above experiments by the crassamentum being reduced to a very small fraction,—one sixth to one twelfth, for instance, of the serum) is not confined to cases of excessive loss of blood, but exists also in an immense proportion, if not the whole, of the reputed nervous cases which present bellows-murmur. *b.* That the murmurs and tremours are partly dependent on the abruptness of the heart's contraction, or, more rigidly speaking, on the velocity with which the blood is propelled in consequence of that abruptness,—a velocity which implies an augmentation of friction, and consequently of arterial vibration. That an increase of the velocity, independent of an increase of the *real* force of the heart's contraction, suffices, under these circumstances, to excite the phenomena, is proved by their existing when the pulse was small and weak, provided it was jerking, and by their increasing when, with the same small and weak pulse, the beat of the heart was accelerated by startling the animal. I conceive the primary moving cause, then, to reside in the heart, and not in the arteries, because the action of the latter was always in exact proportion to that of the former.

To recapitulate: the physical circumstances usually attending inorganic murmurs in the heart and arteries are, 1. attenuation of the blood: 2. unfilled arteries permitting unusual vibration of their walls and a rippling current: 3. a certain velocity of the current occasioned by abrupt contractions of the heart.* I do not see why, when the three preceding causes are in action, a fourth (though I have not seen it noticed by authors) is not brought into co-operation: namely, a disturbance of the current at every point where a branch springs from an arterial trunk. This disturbance necessarily and invariably takes place when rigid tubes are the subject of experiment; and, though the elastic compression of tensely filled arteries in healthy subjects may, in a great measure, prevent it by limiting the vibratory power both of the arterial walls and the blood, such would not be the case in arteries lax from anæmia. I merely submit this as a suggestion, but would meanwhile remark that the arteries in which inorganic or anæmic murmurs are of most common occurrence, happen to be precisely those from which several large branches arise within a small space: namely, the subclavians, carotids, and abdominal aorta.

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* Dr. Corrigan has not attached sufficient weight to the essential circumstance of a certain degree of velocity,—an error into which he appears to have been betrayed by the inaccuracy of the two leading facts on which he founds his theory. 1. In permanent patency of the aorta valves (regurgitation through which, unfils the arteries) he says that the murmur with the *first* sound "*is generally very loud*," and that "the theory of increased velocity will not apply to it, *for the sound exists without it*, and each individual contraction of the ventricle of the heart in this disease is not more rapid, but indeed generally *slower* than in health, whether examined by the stethoscope or by the pulse, which is full and rises *without any jerk under the*

finger" (*Dublin Jour.* vol. x. p. 185-6). Now, I beg to deny the fact that, in this disease, murmur attends the *first* sound. Before me lie nine cases in which it was absent, and I have never known an instance in which it was present, unless the circulation was accelerated or there was more or less obstruction in the aortic orifice. It is with the *second* sound that the murmur takes place—and *invariably*: not "in some cases" only, as he states. The pulse, too, when the regurgitation is at all considerable, is *invariably and preeminently* jerking: it is, par excellence, the pulse of unfilled arteries. 2. Dr. Corrigan states, "that of all the lesions with which bruit de soufflet is connected, it is in contraction of the auricular valves that the sound is most constant" (Ibid. p. 182). I have already shown at p. 78 that the reverse is the case—that the murmur with the *second* sound, to which he refers, is seldom present and always weak, and that the cause of this is simply, deficient velocity and strength of the current.

It may be incidentally remarked that Dr. Corrigan announced "Permanent patency of the mouth of the aorta" as a *new* disease, in the Edin. Med. and Surg. Jour. for 1832: it was, however, described in this work published in December 1831, and I had discovered it several years previously, and taught it, and regurgitations in general, both in St. Bartholomew's Hosp. and La Charité, Paris, as early as 1826. M. Bouillaud, who remarks upon Dr. Corrigan's "*pretended*" new disease, does not say who *was* the discoverer, but seems to allude to M. Guyot, interne to M. Rayer in 1834 (Traité, i. 225). The above dates settle the question.

Though Dr. Corrigan's theory is, as above shown, partly incorrect, and, where true, only an iteration of my own, M. Bouillaud is totally wrong in supposing that his own arguments derived from Dr. Corrigan's error respecting aortic regurgitations, are conclusive against the doctrine that lax arterial walls and a consequently rippling current contribute to the production of arterial murmurs (Traité, i. p. 223). This is one, amongst other reasons presently to be explained, why he has given so singularly unsatisfactory an explanation of the murmurs in question—sometimes confining himself to "données fort générales," and sometimes avowing his total ignorance (Ibid. p. 227-8).

Before closing this division of the subject, I am induced to transcribe the following interesting case in the human subject, related by M. Bouillaud in corroboration of the preceding experiments on dogs. "I was summoned, says he, on Aug. 21, 1834, to see, with the surgeon-major of the 3d Lancers, the son of a *chef d'escadron*, æt. 16, who, after a leech-bite, had lost an enormous quantity of florid blood very probably furnished by a small divided artery. Syncope was imminent: lips and face colourless, eyes turned up and livid, general coldness, long sighing inspirations, pulse exceedingly quick and weak, &c. On listening to the beats of the heart, which were feeble but *very abrupt and frequent* so as scarcely to admit of being counted, I heard a clear, smart bellows-murmur, like the puff with which one blows out a candle. I made the surgeon-major and assistant-major attest the phenomena. I was opinion that the murmur depended solely on the fainting and anæmic state, during which, from the hurried palpitation of the heart, the small column of blood contained in the ventricles was expelled *if not with much force*, at least with a sort of *convulsive rapidity*. I revisited the patient on the following day at the same hour: the hæmorrhage had ceased for more than twenty hours. There did not exist a trace of the murmur, which, according to the surgeon-major's account, had completely disappeared ever since the preceding evening." M. Bouillaud adds that the patient had no organic disease of the heart (Traité, i. p. 180).

We now come to the third branch of the subject: namely, to show—

3. That the explanation applies equally, whatever be the circumstances under which the sounds occur: viz. that there is an increase of friction dependent on a modified motion of the blood.

a. A murmur resembling the slightest and shortest whiff with which we blow out a candle, may be factitiously produced in any considerable artery, as the carotid, subclavian, humeral, femoral or tibial, by slightly pressing it with the end of the finger or with the edge of the stethoscope while we listen. Here, the indentation impressed on the artery causes a local obstruction, which breaks the current, increases the friction, and causes sonorous vibrations both of the blood and the lax walls below. Though the effect takes place in healthy subjects, it is much greater in the anæmic with palpitation and a jerking pulse, and in cases of regurgitation through the aortic valves—a state also attended with unfilled arteries and a remarkably jerking pulse: and the reason why the murmur is louder and the thrill stronger in these two classes of cases, is (in addition to attenuation of the blood in the one class) that the fluid is shot through the stricture with greater velocity, while the vessel, from its unfilled condition, enjoys greater latitude of motion for its own vibrations and those of the enclosed blood. It was probably these circumstances which betrayed Laennec into the erroneous supposition that murmur from compression could only be produced in the hypochondriacal (*De l'Auscult.* ii. p. 443 and 766)—a class of patients who are almost invariably anæmic. I have performed the same experiment of compression, with the same result of murmur and tremour, on the denuded aorta of asses poisoned with woorara (see p. 50). In one instance, I have known compression exercised on the ascending aorta by the consolidated, tubercular edge of the left lung, which took the mould of the vessel and created a murmur whenever the patient lay on her right side, but not otherwise—gravitation in the latter case withdrawing the lung from the vessel. Tumours of any kind, resting upon, and compressing considerable arterial trunks, may occasion murmur in the same way. Of this, tumours resting on the abdominal aorta, receiving its pulsation and simulating aneurism, present the most familiar instances (see *Aneurism of the Abdominal Aorta, Fallacies*). Bouillaud cites a case of a tumour of the left ovary causing murmur in the iliac arteries; and a second, in which a mixed uterine and ovarian tumour, occasioning murmur in the same vessels, was mistaken for the reputed placental murmur of an extra-uterine pregnancy, and led to a fatal cesarian operation (Traité, i. p. 210 and 249). I shall hereafter show that the pregnant uterus is probably to be ranked amongst the tumours in which the attendant murmur is a result of compression (see Auscultation applied to Pregnancy).

Such is the explanation of inorganic arterial murmurs when occurring under the circumstances of compression. It will be shown hereafter that compression and other circumstances occasion a continuous murmur in the veins, and that when arterial and venous murmurs coexist in the same spot, the result is a continuous murmur with arterial augmentations (See Venous Murmurs).

b. Another class in whom the bellows-murmur prevails, consists of those who are under the influence of excessive loss of blood, whether by the lancet or by hæmorrhage in any form. Here, the circumstances are precisely the same as in the dogs above described, and need no further explanation. The jerk and throb of the pulse, even when small and weak, is well known to all who have witnessed a case of uterine hæmorrhage. In cases of active hæmorrhage, as hemoptysis and even epistaxis, I have known the throb to supervene sooner than could be accounted for by the quantity of blood lost. In these cases it appears to be attributable either to the irritable temperament of the individual, or to the fright which seldom fails to be occasioned by the unexpected appearance of blood.

c. By far the largest class of individuals in whom the bellows-murmur is apt to occur, consists of young people, especially females, of delicate, irritable temperament, subject to hysterical and hypochondriacal affections, to nervous palpitation, and in many cases to hæmorrhages. From careful observation during the last ten years, and from examining the blood of such patients when opportunity offered, I feel assured that the whole, or nearly so, whether male or female, are affected with more or less anæmia, that is, a deficiency in the quantity of the blood, or in the proportion of its fibrine and red globules, or in both,—constituting in females the state inappropriately denominated chlorosis. This state is not incompatible with a full, but flabby habit of body, and in such subjects especially I have seen the hæmorrhagic tendency prevail to an extraordinary degree, the flux occurring successively from all the mucous membranes. The seat of the murmur, when cardiac, is in the aortic orifice and with the first sound (For the diagnosis, see Physical Signs of Valvular Disease), and, when arterial, in the carotid, subclavian, brachial, crural and abdominal aorta more especially, though any other large trunks may yield it. The murmur is not constant, but occasional, coming on whenever the circulation is excited, and, for exciting it, the most trivial causes, as Laennec has observed, are sufficient. I have seen a single cough, or a full inspiration, or a little flatulence, produce the effect for a few beats only; while the act of turning in bed, of rising suddenly, of being startled by any noise, has occasioned it for several minutes. An emotion of grief or pleasure will sometimes produce a more considerable and permanent effect. I have often been assured by patients that the momentary flash of an idea across the mind, has sufficed instantly to excite a violent fit of palpitation, and that this has recurred several times a day, whenever the same idea has presented itself. The high nervous irritability of the anæmic dogs (see p. 101) was precisely parallel. That, under so irritable a state of the nervous system, the heart should contract with spasmodic abruptness, might be anticipated; and what theory points out, experience proves; for the jerking pulse and beat of the heart of a chlorotic patient in a state of nervous agitation, are too well known to require further comment. Sometimes, when the nervous excitement is excessive, a violent throbbing is perceptible, as in the dogs, over the whole body, and the bellows-murmur and thrill are distinct in every considerable arterial trunk (*Laennec*, ii. 442). When such is the case, the anxiety and distress of the patient are extreme, and his situation is not exempt from danger, though, as Laennec truly observes, death is rare when the symptoms are independent of organic disease of the heart.

It may here be remarked that the jerking pulse of anæmia differs from the inflammatory pulse in neither being full, strong nor hard; while the inflammatory pulse has neither the jerk, thrill nor bellows-murmur of the anæmic pulse. When the latter is modified by inflammation, it acquires a little strength which converts it into what is denominated the sharp pulse—the inflammatory pulse of feeble subjects. These distinctions, which to the inexperienced may appear refined in description, are perfectly familiar to practical men; and it is of great importance to the young practitioner that he make himself intimately acquainted with them, as such knowledge will not only facilitate his diagnosis, but prevent the unnecessary and often, in anæmic nervous cases, pernicious abstraction of blood for imaginary fever or inflammation.

d. In *arterial varix*, a variety of aneurism by anastomosis graphically described by M. G. Brechet (Mémoires de l'Acad. Royal de Med. tom. iii. p. 136-174. 1833), bellows-murmur and thrill exist in a high degree. The tumour in Brechet's case was soft and spongy to the feel; it swelled with each systole of the heart, the cells being dilated, the skin rendered tense and the volume increased; the skin, moreover, was so thin as to show a net-work of cells, to which the blood imparted a dark-red colour every time it was injected by the ventricular contractions. Pressure being made on the common carotid, the whole tumour became pale and small, and, on withdrawing the pressure, it rose again, with bruissement, in from eight to sixteen pulsations. The tumour was found to consist of arteries dilated, tortuous and forming a web; and the capillary vessels, venous as well as arterial, were found greatly dilated: the coats of the arteries were thin, soft and flaccid. Now, friction and vibration must in such cases necessarily exist in a preeminent degree; for the current is broken by an infinity of anfractuosities, and the softness of the tumour gives the same laxity to the arterial walls as they derive in anæmia from the unfilled state of the vessels. I entertain no doubt, however, that the murmur and thrill would be increased and perhaps converted into a continuous murmur by anæmia; such tumours, therefore, form no exceptions to the general rule, that murmur and thrill are referable to increased friction from modified motion of the blood.

Musical Murmurs have been described by Laennec, and still more elaborately by Bouillaud, as occurring in the arteries; nor has their doctrine, to my knowledge, been hitherto controverted. I feel assured, however, that this phenomenon, no less than the continuous murmur, is seated not in the arteries, but in the veins. For, first, the musical murmur is always continuous; whereas, the arterial bellows-murmur is always intermittent, accompanying the ventricular systole and ceasing during the diastole;—a fact of which the authors alluded to were not aware, in consequence of their beiug strangers to the existence of the venous murmur, whence they did not distinguish it from the arterial. Secondly, I have never met with the musical murmur except in cases where the continuous venous murmur existed. The consideration of the musical murmur, therefore, falls under the next section, where the direct proofs will be given that it is seated in the veins.

SECTION V.

VENOUS OR CONTINUOUS MURMUR, HUM AND MUSICAL NOTES.

Laennec was the first that noticed the continuous murmur, though he mistook its cause. "In some rare cases," says he, "the bellows-murmur changes, in the carotid especially and even in the heart, into a continuous murmur analogous to that of the sea, or that which we hear on bringing a large univalve sea-shell near the ear. The *saccade* or shock of the arterial diastole is then no longer distinguishable, or *only very feebly.* Sometimes this continuous murmur exists in one of the carotids or subclavians, while the corresponding artery yields the ordinary bellows-murmur, that is to say, rythmic and isochronous with the arterial diastole. Most commonly, bellows-murmur (here Laennec means arterial and cardiac,) is exactly circumscribed by the calibre of the artery or by the capacity of a ventricle. *In other cases, on the contrary* (here he unconsciously describes venous murmur,) *it is diffuse, and seems to take place in a space far more extensive* (VASTE) *than the artery or the heart, of which we no longer perceive either the impulse or form*" (De l'Auscult. ii. 422). This continuous murmur, Laennec ascribes, as usual, to nervous spasm of the heart and arteries. He makes a distinction (which I shall hereafter show to be incorrect) between it and the continuous rumble of *bruit musculaire* accidentally excited in a muscle contiguous to the artery under examination; yet he adds, as if from some misgivings of his first explanation, "I have sometimes suspected that the continuous murmur might depend on spasmodic contraction of the sterno-mastoid and platysma myoides. I have sometimes, but not always, *made it cease by relaxing these muscles*" (Ibid. p. 447).

M. Bouillaud, following Laennec, refers the continuous murmur to the arteries, and connects it with the constitutional cause which I had previously pointed out, namely, the anæmic state;

but he confesses that his arterial theory is totally insufficient to account for all the circumstances attending the production of the murmur.

Upwards of ten years ago I had come to the conclusion that the continuous murmur was unconnected with the heart and arteries; since its diffuseness, and in some cases its singular loudness, were incompatible with such an idea: I was equally incredulous of its being an ordinary bruit musculaire; since its loudness sometimes incomparably exceeded the most intense muscular sound, and I had frequently found it excited by mere pressure, which will not give rise to muscular sound. In this uncertainty as to its cause, I continued to collect cases and make observations on the continuous murmur, but had not leisure to concentrate particular attention on the subject.

Dr. Ward of Birmingham, in March 1837, solved the enigma. He ascribed the murmur to the current of blood in the veins (Med. Gaz. vol. xx. p. 7.), and made ingenious attempts to unravel the perplexities of M. Bouillaud. Since that time, I have made observations on a vast number of cases, and have found Dr. Ward's solution correct in the main, but defective in many of the particulars—especially in his explanations of several of the phenomena described by M. Bouillaud, which are themselves incorrect. As my limits do not permit me to analyse the able researches of these gentlemen, I shall describe the venous or continuous murmur such as I have found it, and shall explain the concomitant circumstances as I proceed.

The venous murmur is on a much lower key than the arterial bellows-murmur; for, while the latter is often as high as the note produced by whispering the letter *r* and seldom lower than *au*, the venous murmur is usually as low as *who*. This sound, indeed, offers the most complete and ready imitation of the phenomenon with which I am acquainted. The hollow sound of a large incessant forge-bellows also imitates it very closely. When there is no considerable arterial throbbing, the venous murmur maintains an even tenour, "the *saccade* or shock of the arterial diastole, to use Laennec's words, being no longer distinguishable, or only very feebly." But when the arterial throb is considerable, the murmur experiences augmentations corresponding with each arterial diastole or pulse, precisely as when the handle of the forge-bellows is depressed, or when we force the breath while whispering a continuous *who.* These augmentations are nothing more than the superaddition of the arterial whiff to the venous murmur. I have constantly been able to distinguish the former *through* the latter by its higher key, and also by its being closely restricted to the tract of the artery, whereas the venous murmur sounds wide and diffuse. The frequent coexistence of this arterial murmur has led M. Bouillaud into the error of imagining that augmentations are one of the inseparable and essential characters of the continuous murmur, which is far from being the case. In some instances, I have observed that the arterial whiff seems momentarily to check the venous murmur, and I think it probable that, by compressing the vein enclosed within the same sheath, it actually does so; for the art of swallowing certainly produces this effect, in consequence of the internal jugular vein being compressed by the elevated os hyoides. The result is, that a sound is produced conveying to the mind the impression of a current rushing forward, and then back again—which is, indeed, an expression of the fact, except that the currents are in different vessels. I suspect that it is this phenomenon which has led M. Bouillaud to denominate the continuous murmur "bruit de soufflet continu ou à double courant."

The venous murmur, whether simple, humming or whistling, (for I shall presently show that it presents these two latter characters), increases and diminishes, or suddenly ceases and as suddenly returns, from causes which appeared capricious and inexplicable to M. Bouillaud, in consequence of his erroneously ascribing the murmurs to the arteries, but which are perfectly simple and explicable on the correct view, and constitute, indeed, so many proofs that the phenomena are seated in the veins.

a. When the vein under examination is very superficial—merely subcutaneous, as the external jugular, very light pressure with the stethoscope will increase the murmur by partially contracting the calibre of the vessel; but if the vein be obliterated by laying the point of the finger lightly upon it above the stethoscope, or by depressing the upper edge of the stethoscope, the murmur instantly ceases. It is seldom, and only in the most marked cases, that murmur can be detected in the external jugular veins. That of the internal jugular may easily be mistaken for it by novices.

b. Strong pressure with the stethoscope, sufficient to obliterate a subcutaneous vein, as the external jugular, instead of suspending the murmur, swells it *gradually* to a surprising degree of intensity and diffuseness, like blowing the word *who* with great force, mixed up with which sound I have frequently heard humming, cooing and whistling notes, appearing to proceed from several veins at once, which I shall hereafter show to be the case.

It was this extremely loud murmur which first led me to suspect that the continuous murmur of Laennec was neither seated in the arteries, nor occasioned by ordinary bruit musculaire. The loud murmur in question, though noticed by Bouillaud, (ii. p. 214), has not hitherto been explained. I have found it to depend simply on the circumstance of the compressed veins being more deeply or remotely seated, whence, though the depression of the stethoscope is forcible, the pressure exercised on the veins is only moderate—such as merely to contract, without obliterating their calibres. It is not always necessary that the stethoscope should be placed directly over the vein, the same effect being produced by its displacing parts, and thus creating pressure laterally. These facts are easily put to the test on the internal jugular vein. This vessel runs, as depicted in Fig. 1, in front of the carotid artery, along the anterior margin of the sterno-mastoid muscle from the angle of the jaw to below the middle of the neck, and is separated from the surface merely by the integuments and platysma myoides. If the stethoscope be placed on the posterior side of the sterno-mastoid muscle while the face is averted but the neck kept perfectly erect and the chin well raised, firm pressure gradually develops the loudest continuous murmur that it is possible to create in the individual under operation, and it is "cæteris paribus" louder in proportion as the subject is more anæmic and excitable. If the point of a finger be now nicely dropped on the internal jugular vein in any part of its course, so as to obliterate the vessel, yet without obliterating the carotid, the loud murmur instantly ceases, and nothing is heard but a dull, obscure rumbling, seated in smaller veins. This may sometimes be further diminished by obliterating the external jugular, but it cannot always be completely annihilated, because it occupies deep-seated veins beyond the reach of sufficient compression. It is occasionally mixed up with puny humming and whistling notes. If the finger be now raised again from the internal jugular, the torrent rushes down and restores the original loud murmur almost as promptly as when the finger is raised from the hole of a wind instrument. By alternately raising and depressing the finger, the most sceptical may soon convince himself that the seat of the murmur is really in the vein. One or two precautions are requisite. If the neck be displaced from the perpendicular, the sterno-mastoid muscle is apt to be put so much on the stretch as to obliterate the internal jugular and suspend the murmur. Again, if the stethoscope, placed behind the sterno-mastoid, press that muscle too much forward, it will obliterate the internal jugular. Again, if the skin be stretched across the neck, under the stethoscope, the tension will increase the murmur in most; but in a few, whose internal jugular is very superficial, it will obliterate the vessel and suspend the murmur.

I have met with the remarkably loud murmur in question, not only in the internal jugular veins, but also on the abdomen,—principally, I think, over the right side of the umbilical and epigastric regions, where lie, in addition to the internal mammary veins, the great converging branches of the vena portæ and the renal veins. Very heavy pressure with the stethoscope is requisite to excite the murmur on the abdomen. I have generally met with it accidentally, when exploring pregnancy or supposed aneurisms of the abdominal aorta, and the instances of its occurrence to me may have amounted to fifteen or twenty. I have likewise met with a less degree of the continuous murmur in the iliac and hypogastric regions, its seat probably being, the external iliac and the epigastric veins; and I have ascertained by researches directed specifically to the subject, that both arterial and venous murmurs may be excited by pressure on the several vessels enumerated, in the majority of anæmic and excitable subjects with a quick pulse, whether they are emaciated or otherwise. The further consideration of abdominal murmurs is reserved for the Chapter on Auscultation applied to Pregnancy.

c. It was stated under the preceding head, that the loud murmur there considered, swelled *gradually:* that is, when the stethoscope is first applied, it is inaudible or slight; but after a few seconds it begins to swell, and this progressively increases till, in the course of from ten to twenty or thirty seconds, it attains its maximum of intensity. I was some time before I could unravel the cause of this *gradual* swell, but I at length discovered that it depended on the simple circumstance of congestion of the veins *above* the part compressed, by which congestion, or, in other words, increased *vis a tergo*, the current through the compressed portion of vein is rendered stronger and more rapid: therefore, as the congestion takes place gradually, the swell of the murmur is gradual in the same proportion. I was led to this discovery by observing that the murmur of the *external* jugular did not attain its maximum of loudness and become musical in a certain case, till the portion above the stethoscope became exceedingly tumid.

I have further observed that the loud murmur of the internal jugulars becomes louder during inspiration,—especially about its end, and weaker during expiration. I ascribe it to expansion of the lungs opening a freer channel to the blood on the right side of the heart and thus depleting the jugular veins below the part compressed,—circumstances which, by creating a tendency to a vacuum, would accelerate the current through the compressed portion, while the unfilled vein below would be in a state favourable to the ripple of its current and the vibration of its walls. During expiration, the circumstances are reversed and the opposite effect is therefore the result. I have noticed that when an anæmic person becomes faint from standing long under examination, the murmur, previously loud and constant, becomes extinct except during the inspirations. This evidently proceeds from a deficient afflux of blood to the head, whence there is not a sonorous current down the veins, except when it is favoured by the suction of inspiration. For the latter reason, the murmur exists during inspiration only, in those who barely exhibit the phenomenon at all,—for instance, the convalescents from anæmia.

d. In the subdivision *b.* it was stated that, in order to produce the murmur of the internal jugular in perfection, it was necessary to avert the face while the neck was kept perpendicular and the chin well raised. The mode in which this position acts is, it my opinion, by placing the vessel in a state of moderate tension, which is favourable to the vibration of its walls, and also increases the sonorous effect of pressure accidentally exercised on it at any particular point, as, for instance, by the sterno-mastoid muscle where it crosses in front of the vein. Accordingly, when the head is restored to its natural position, or is depressed, the vein is relaxed and the murmur ceases or greatly diminishes. A beautiful illustration of the effect of tension of a vein in producing murmur, is presented by the following case of one of my present patients at St. George's Hospital, January 18, 1839. Phebe James, æt. 13, has consolidation, contraction, inexpansibility and universal dulness on percussion of the right lung, connected with dilatation of the bronchi, yielding singularly loud pectoriloquy and gargouillement over the upper and middle lobes. No phthisical emaciation or perspirations though copious expectoration has existed constantly for three years. The left lung is greatly hypertrophous and universally presents puerile respiration. By these circumstances the heart is displaced so completely to the right side that the ascending aorta can be felt to beat between the 2nd and 3rd right ribs, about one-and-a-half or two inches from the sternum. In this spot, the two sounds of the heart, especially the second, are loud and perfectly exempt from murmur; but, on listening at the edge of the sternum, *a continuous murmur with augmentations corresponding to the pulse, becomes audible and may be traced with increasing loudness along the tract of the vena innominata across the sternum to the opposite side.* Here, I can only suppose that the stretched state of the vein renders it more susceptible of vibration and of the sonorous effect of accidental indentation from parts which it crosses. There was venous murmur in the jugulars also, the patient being anæmic. (The notes of the case were taken for me by Messrs. Pollock and Mayor, students of St. George's.)

e. M. Bouillaud states, and Dr. Ward acquiesces, that the continuous murmur in the jugular veins ceases or diminishes when the larynx is pushed to the opposite side; whence M. Bouillaud imagines that the larynx and trachæa constitute a sort of sounding board to the carotid arteries, in which he supposes the murmur to be generated. The whole is a mistake. When the murmur ceases, it is most frequently in consequence of the thumb being inadvertently placed upon and obliterating the internal jugular; but it is sometimes in consequence of the tension of the skin being so great as to obliterate the internal jugular either by compressing it against the anterior margin of the sterno-mastoid or by depressing this muscle upon the vein at the point where they cross. When these several circumstances are avoided, and the larynx is pushed aside with moderate force, the murmur, so far from being diminished, is increased; for I have found that moderate transverse tension of the skin generally produces this effect—perhaps by giving steadiness to all the parts beneath, which is favourable to vibration and to the production of local indentations on veins by contiguous muscles or by external agents.

As the respiratory murmur simulates the venous murmur, learners should request the patient to hold his breath.

f. M. Donné and M. Bouillaud, who quotes him, state that any considerable corporeal efforts immediately suspend the continuous murmur. I have not found this to be the case, and I imagine that those gentlemen have been deceived by inadvertently allowing the head to fall into some of the positions above described as unfavourable to the production of the murmur.

g. Laennec thought the continuous murmur to be louder and more frequent on the right of the neck than on the left, while M. Bouillaud has come to the opposite conclusion. I have scarcely ever found it on one side without being able to discover it on the other, but I think it is generally louder on the right side,—probably in consequence of the course of the vein to the heart being straighter, and therefore more favourable to hydrostatic pressure.

h. After the explanations above offered, it is evident that there is nothing which does, or can imitate the venous murmur but *bruit musculaire*. That, however, it is not muscular sound, is to me certain for the following reasons: 1. *Bruit musculaire* can be created by muscular action in healthy subjects; but the murmur in question cannot be excited, or only very slightly, except in anæmic or naturally thin-blooded subjects. 2. I have placed the end of one finger on the external jugular vein, and the end of another on the internal, above the stethoscope: by depressing both I could wholly and instantly suspend the murmur, and by raising either, I could reproduce it at pleasure in either vein. Now, a murmur from *bruit musculaire* would not be suspended by depressing the point of a finger on a few fibres of a muscle. To succeed with this experiment, the edge of the stethoscope must only lightly touch the side of the external jugular, the murmur of which can with difficulty be excited, and only in marked cases.

It is quite manifest to me that Laennec actually did mistake the venous murmur for bruit musculaire. No one will, I think, doubt this who compares the following passage from that author with all that precedes. "An inexperienced observer might believe in the existence of a bruit de soufflet (of an artery) without its being real, when bruit musculaire is accidentally developed in a muscle near the artery explored. This happens especially in the carotid, *in some persons labouring under more or less considerable nervous* (*and anæmic?*) *agitation*. If, when the patient is seated, we make him *incline his head to the left side*, so that it be sustained solely by the right sterno-mastoid muscle, this muscle then takes on the mode of contraction which yields the bruit de rotation (rumbling of distant wheels—really, the venous murmur). Further, the carotid, swelling at each diastole, impresses a slight shock on the muscle, the rumble of which then appears intermittent like the arterial whiff, and from that circumstance strongly resembles the bellows-murmur (in arteries); but with a little attention it may be perceived that the rumble is *remittent* rather than intermittent. (Here Laennec evidently describes the continuous venous murmur with its augmentations from the arterial whiff.) We should not, however, trust the position of the patient; for, *on causing him to make a very slight movement of the head towards the side under examination*, or on supporting the head, were it only with one finger, we instantly suspend the bruit musculaire. I have sometimes suspected that the CONTINUOUS MURMUR, of which I have spoken above, might also depend on a spasmodic contraction of the sterno-mastoid and platysond myoides. I have sometimes, but not always, made it cease *on putting these muscles off the stretch*" (De l'Auscult. ii. p. 447).

Dr. C. Williams has fallen into the same mistake as Laennec; for, as shown above at p. 52, he has quoted the very same circumstances as exemplifying what he considers to be the type of bruit musculaire. Thus, in so strenuously ascribing the first sound of the heart to bruit musculaire for the last ten years, he has been ascribing it to a non-entity; for I am strongly inclined to believe that, not only in the neck but universally, bruit musculaire will hereafter be found to be nothing more than a venous murmur excited by the muscular contraction. This idea does not originate with myself. I heard it suggested many years ago, but cannot recollect by whom.

I have never been able to feel a thrill in the external or internal jugular or any other vein, but I have felt it over the arteries of a part yielding venous murmur: it may be a question, therefore, whether the current in veins is strong enough to produce a thrill, and whether, when it exists over veins, it is not referable to the concomitant arteries. Laennec seems to have observed a diffuse tremour. "Sometimes, on the contrary, and particularly in the carotid, the tremour is much more extensive than the diameter of the artery and appears to be more superficial. The tremour of the carotid is sometimes perceptible over a space two inches wide on the sides of the neck, and then it is more perceptible in proportion as the extremities of the fingers are lightly pressed on it. This tremour then appears to be continuous and the arterial shock is not at all felt."

Musical Venous Murmurs. It has been stated (p. 108) that musical notes in the blood-vessels are not seated in the arteries, as Laennec, Bouillaud, &c. imagine, but in the veins. The proofs of this are now to be offered.

By the adroit management of pressure with the stethoscope over or near large veins, the venous murmur may often be raised, by a gradual swell, into a more or less musical hum, such as is yielded by a child's humming-top. I propose to denominate this the *Venous Hum;* for without being unnecessarily squeamish, I think that this is not only a rather more euphonous epithet, but more intelligible than *noise of the devil,* by which term, derived from a plaything known to few, M. Bouillaud has designated the hum in question.

Sometimes, again, either with or without hum and with much or little murmur, we hear a more perfect, continuous musical note, like a delicate whistle produced by the lips—or by the wind traversing a key-hole or crevice, or, in some instances, like the singing of a kettle, or the song of a musquito fly.

In proof that these musical notes are generated in the veins, and not in the arteries, the following, out of many other cases of the same kind, may be adduced. On a large bronchocele, in an anæmic boy of æt. 15, a musical note like the slightest whistle, and a continuous, dull, rumbling murmur, were both suspended whenever I pressed my finger transversely on the neck, above the tumour, so as, without compressing the carotid artery, to obliterate the superficial veins descending over the surface of the tumour. A still more conclusive proof is, that in a patient now before me, I can at pleasure create a musical note like the song of a fly (running alternately or a tone and a semitone above), in the external jugular vein, by delicate and well-managed pressure on that vessel with the stethoscope; but the note stops the instant that I place the point of my finger on the vein above. Further, by heavier pressure with the stethoscope, I can at pleasure develope an exceedingly loud venous murmur in the *internal* jugular, together with a beautiful musical note like the singing of a kettle, which runs *continuously* on two notes, the one a perfect major third above the other. The transition is marked by the arterial pulsations, and the two notes generally alternate at each pulsation; but sometimes the lower note persists during three or four consecutive pulsations, being merely augmented by each. Further, I can create the musical notes, not only by pressure with the stethoscope but also with my finger. For on lightening the pressure with the stethoscope, so as to extinguish the musical note but leave the ordinary loud venous murmur, I can reproduce the musical note by pressure with the point of one finger over the internal jugular vein in front of the sterno-mastoid muscle, below the jaw, the chin being raised and averted; and, by increasing or diminishing the degree of pressure (always without obliterating the vein) I can raise or depress the note at pleasure, sometimes a whole tone, sometimes only half. On depressing the finger with sufficient force to obliterate the vein, *without obliterating the carotid artery*, the continuous murmur, together with the musical notes, instantly cease: which is a conclusive proof that both are seated in the vein. I have found very few cases in which I could make this experiment succeed so well.

The internal and external carotid are not the only veins of the neck in which the musical murmur can be produced. I have found in several cases that, when I obliterated these vessels and suspended their venous murmur, puny musical notes and a feeble obscure rumble could be developed by well-managed pressure with the stethoscope on the intermediate space. Their seat was probably in the smaller and deeper-seated veins corresponding with the branches of the subclavian. The musical note was so weak as to be drowned by the loud murmur of the internal jugular whenever the finger was removed from this vessel.

Stretching the skin transversely under the stethoscope, often favours the conversion of an ordinary venous murmur into a musical note, the cause being, an increase in the fineness and rapidity of the vibrations.

Musical notes are much more difficult to produce when patients are recovering from anæmia. They appear to me to be most common in a class of young females of what is called the "phlegmatic temperament," that is, with a lax, flabby muscular system, large blood-vessels, thin arterial coats, proclivity to hæmorrhages, often a rather full habit, and whose blood, even during health, contains less than the average of fibrine and red globules.

In all cases, the production of the musical murmur requires patience and adroitness; as the auscultator may be foiled again and again and as if capriciously, by an unhappy position of the stethoscope, a deficiency or excess of pressure, or a movement of the head relaxing the veins or the skin.

Mr. Mayo and Mr. Wheatstone lately did me the favour to examine three patients and verify most of the preceding facts.

If the above account be now compared with Laennec's description of musical notes existing, as he erroneously imagines, in the arteries, it will be manifest that we are each alluding to the same phenomenon, and, therefore, that its seat is in the veins.

"The arterial bellows-murmur," says this acute observer, "frequently passes (especially at times when the patient is more agitated than usual by any cause whatever) into a whistling analogous to that of the wind blowing through a key-hole, or to the resonance of a metallic chord which vibrates long after having been touched. The resonance of thediapason which issued to tune keyed instruments, may also be perfectly imitated by the whistling noise of arteries. These sounds, always feeble, are nevertheless very appreciable, and we may easily find the note that they represent in reference to a given key-note or diapason: further, in some cases, rare it is true, the sound ascends or descends by intervals of a tone or half a tone, as if the artery had become a vibrating chord on which a musician produced successively two or three notes by advancing or withdrawing his finger" (De l'Auscult. ii. p. 423). Laennec describes one case in which the air ascended and descended on three notes constituting a major third, except that the upper note was slightly too flat. The "transition from one note to the other," says he, "was occasioned by each arterial diastole," and the lowest note or *tonic* was sometimes sustained for a variable period, the arterial diastole then occasioning merely an increased intensity of the sound, which decreased during the systole. This same variation in intensity occurs, he says, when the musical sound is confined to a single note. Occasionally, the sound fails during the systole and is only heard during the diastole. The music may from time to time cease suddenly and be replaced by an ordinary bellows-murmur, and, when the circulation becomes calm, it ceases altogether. Such is the substance of Laennec's account of musical murmurs.

Some have experienced a difficulty in conceiving how an ordinary murmur should be converted into a musical note. It has already been stated, in reference to musical murmurs of the valves of the heart, that there is but a shade of difference in the mechanism by which we make the lips produce a blow or a whistle, the latter depending on the happy and steady adaptation of the size of the aperture to the strength of the current. A more apposite illustration may be drawn from the child's toy called the humming-top—a hollow top with a hole in the side, and spun by drawing a string twisted round the upper end of its axis. When the string is first drawn, it dances about with a humming murmur; but when it becomes steady and, in the child's phrase, "goes to sleep," the hum is gradually converted into a clear and agreeable musical tone, which dies away again into a feebler hum as the rotation becomes too weak to maintain it. Here, a certain proportion between the velocity of the current of air and the size of the aperture, aided by the steadiness of the instrument, gives rise to fine vibrations in the wood calculated to be musical.

A step further brings us to wind instruments. Every expert flute-player knows that the purity, richness and variety of his tone depends, not on the brute strength with which he blows, but on a happy adaptation of the volume and strength of the current of air to the size of the embouchure, which he increases or diminishes at pleasure by withdrawing or advancing his lips over it: also, that steadiness is indispensable, the slightest quiver of the lip or movement of the instrument being fatal to the tone: further, that he can "force" a "reedy" tone on the lower notes to a surprising degree of loudness; whereas, one who has not the art of producing a reedy tone can never produce even loudness, though he blow with ten times the force: finally, that an exquisite note, scarcely louder than a whisper, may be elicited by a current of air so fine and feeble as to be inaudible even to the player.

Now, why is not a vein in circumstances analogous to those of a flute? We are certain that it can produce sonorous vibrations, for we hear the murmur. Why may not the several circumstances be so happily adjusted as to render the vibrations musical? Why may not the note admit of being "forced" as we actually find it to be by the arterial diastole? Why, if the relation of circumstances continues correct, may not the note persist in any degree of delicacy, during the feebler venous current connected with the arterial systole and subsequent repose? Why may not the note be suddenly arrested (as we actually find it to be), when the adjustment of circumstances is disturbed by accidental extrinsic causes, as an undue increase or diminution of pressure, a movement of the head, &c.? And why should not the music permanently cease, when, from diminution of nervous and arterial excitement, the circulation becomes too calm to produce the requisite vibrations? If these considerations be true, the phenomenon is explained so far as it lies within the province of the physician: it is for the professor of acoustics to develop the ultimate laws by which given vibrations produce musical, rather than murmuring sounds.

The constitutional causes of the venous murmur, hum, and whistle are exactly the same as those of the arterial bellows-murmur. Laennec had stated this to be "very common in hypochondriacs and hysterical women; also in delicate irritable young people subject to hæmorrhages;" but it was not understood by him or others that *anæmia* was an essential character in these, and all other cases yielding inorganic murmurs, until this fact was pointed out in the first edition of the present work in 1831, in connexion with the experiments on dogs by repeated blood-letting (see back p. 101). Since that period, I have not met with any instance of the venous murmur in a marked degree, in which anæmia was absent. I have to-day found it in the highest perfection in eight out of a hundred of my female hospital patients, and slightly in five more. The whole were anæmic. Further, I have invariably found that the murmurs, &c. gradually disappeared in proportion as the anæmic state was removed by iron, aloetic aperients, animal food and fresh air. If confirmation of facts so easy to be proved were wanting, I might cite the more recent researches of M. Bouillaud, who was acquainted with, and quotes my experiments on dogs (Traité, i. p. 182), who followed up the same train of investigations and arrived at identical conclusions. Thus, he says, "In all the patients in whom I have hitherto met with the *noise of the devil* and its various shades, what *is* the most remarkable general and constitutional condition, if it is not a state of real *anæmia*, or at least a state of the blood in which *the serous portion predominates over the red particles and fibrine*—a state which the reader will permit me to designate by the term of *hydræmia* (watery blood) to avoid circumlocutions? If this state is, as must be presumed, one of the principal causes, if not the sole cause, of the *noise of the devil*, is it not probable that this noise should exist in individuals who, in consequence of accidental losses of blood or of copious blood-lettings and a diet almost wholly aqueous, have fallen into a temporary, but real state of anæmia or *hydræmia?*" (*Traité*, i. p. 22.) This is an almost verbal confirmation of the passage above alluded to, at p. 75 of the first edit. of this work and at p. 102 of the present.

It must be understood that, under the term anæmia, I comprise all patients, whether male or female, whose countenance exhibits an exsanguine paleness, whose blood is thin and serous, and who, in addition, have usually palpitation and shortness of breath on exertion, weakness and aching of the limbs and back, lassitude, constipation, anorexia—usually with disgust at animal food, and, if females, amenorrhœa or menorrhagia—though these are not essential characters. I have, however, frequently found a slight degree of the venous murmur in healthy persons with naturally thin blood. The term *chlorosis*, (from χλωρς, viridis, green or greenish yellow,) should be discarded; as habit has restricted it to females, and connected it with a doubly false theory; since chlorosis is not *necessarily* attended either with a "green and yellow" colour or with suppressed catamenia.

The limits of the present work will not admit of the citation of cases of inorganic murmurs, though upwards of fifty are in my journals, and I feel sure that I have seen three or four times that number. Nothing, indeed, but weariness of noticing the same thing has prevented the number from being much larger; for the venous murmur exists more or less in almost every case of anæmia, and of such cases, my hospital out-patients alone afford upwards of 300 per annum.

The knowledge of the venous murmur is a useful accession to medical science; for it not only constitutes a criterion of the anæmic state and of the degree of attenuation of the blood, but facilitates the diagnosis of organic from inorganic murmurs of the valves, by affording strong presumptions of the latter.

SECTION VI.

PURRING TREMOUR OR THRILL OF THE HEART AND ARTERIES.

Though all the circumstances of this phenomenon have been noticed in connexion with the various murmurs which it accompanies, yet it may be convenient to collect them into a brief synopsis. Tremour arises from the vibrations into which the blood and surrounding solids are thrown during the passage of the fluid through an obstructed orifice or through imperfectly filled or rough vessels.

1. *In the heart.* Tremour is produced by contraction of the semilunar valves or of their respective orifices; but it is rarely felt, because the sternum is interposed: if, however, the heart is displaced from beneath the sternum by hydrothorax, empyœma,

(even circumscribed,) emphysema, encephaloid or other tumours, &c. the tremour may then become perceptible in the region of the semilunar valves. A tremour of the pulmonary artery, however, whether resulting from contraction of its orifice or from dilatation or ossification of the artery itself, may often be rendered perceptible by no other displacement than that resulting from an inclination to the left side while the patient lies in the horizontal position; in consequence of which, the upper part of the vessel is brought by gravitation between the cartilages of the 2nd and 3rd ribs, where the tremour can then be felt. In dilatation of the pulmonary artery the tremour is very distinct at this point, though the patient be erect (cases of Wetherley, L. P., and James). A regurgitating current through the semilunar valves produces less tremour than a direct one, because it is weaker. A current, both direct and regurgitating, out of the aorta through an aneurism into the mouth of the right ventricle, produced a strong tremour in the case of Mitchell, whose heart was also displaced.

Regurgitation through the mitral valve I have observed to be beyond comparison the most frequent cause of tremour in the heart, since the current is strong, and the tremour admits of being felt through the costal interspaces. It is very rare in the tricuspid valve, because the refluent current is weaker, the valve itself is seldom diseased, and the situation is more covered by the sternum. I have never known the direct current through either auricular valve to produce a tremour, the stream, I presume, being too weak.

A strong tremour is produced in pericarditis by the attrition of dry lymph, or the agitation of a small quantity of serum between layers of rough lymph (case of Jones).

I have never known tremour to exist in the heart independent of organic causes.

2. *In the arteries.* Considerable contraction of the aortic valves will occasionally propagate a tremour as far as the carotid and subclavian arteries during palpitation, or even during a calm state, provided there is great hypertrophy with dilatation; but it is rarely, if ever, propagated so far as the radials, unless the effect is favoured by an unfilled state of the arteries from anæmia or aortic regurgitation. Roughness or dilatation of the ascending aorta

and arch occasions tremour in the vessel itself and also in the carotids and subclavians, even during a state of calm: during palpitation, especially if there be hypertrophy, it may be propagated in a slight degree as far as the radials.

Aortic regurgitation, by unfilling the arteries (a state highly favourable to their vibration), may, during palpitation, propagate a tremour as far as the radials or still more remote arteries.

3. *Inorganic tremours.* Anæmia, on the same principle of unfilling the arteries, favoured also by the watery state of the blood, may, during nervous excitement, give rise to a thrill in any or all of the more considerable arteries, but especially in the carotids, subclavians, brachials and crurals. I at present attend a highly anæmic young lady with a pulse of 150, in whom it exists in a most marked degree in the radial artery. The co-existence of anæmia and aortic regurgitation augments the effect, and both, or either favours the operation of the organic causes.

As tremour has the same origin as bellows, musical and other murmurs, it is always accompanied by them; but, as it requires a greater degree of vibration for its sensible development, they may exist without being accompanied by it.

CHAPTER V.

AUSCULTATION APPLIED TO PREGNANCY.

M. Mayor, of Geneva, was the first who applied auscultation to the study of pregnancy by discovering, before the year 1818, that the beats of the fœtal heart could be heard, and distinguished from those of the mother, by applying the ear to the abdomen (*Bibliotheque Universelle*, tom. ix. for Nov. 1818: see note by the editor to M. Percy's report to the Institute on *Mediate Auscultation*). As M. Mayor never afterwards published, Laennec infers that he did not extend his observations beyond the above remarks. In 1822, M. Kergaradec, apparently unacquainted with M. Mayor's discovery, published his memoire on *Auscultation applied to the Study of Pregnancy*, and his results have subsequently been more or less verified, corrected, and extended by innumerable auscultators, including, especially, M. Laennec in 1826, Dr. Ferguson,* Dr. Kennedy,† M. P. Dubois in 1832,‡ and M. Bouillaud in 1835.

The two signs which are considered to indicate a living fœtus in utero, are, 1. The double sound of the fœtal heart; 2. A murmur usually called utero-placental.

1. *The double sound of the fœtal heart.* Writers, including Laennec (ii. 466), have said so much on the difficulty of hearing the fœtal beat, that the following preliminary directions, by which that difficulty may in a great measure be obviated, will not, perhaps, be unacceptable to the novice.

It is useless to attempt an examination in the erect position and through the ordinary dress. The stays should invariably be taken off, as their compression above tightens the walls of the abdomen below. The patient should be in bed, on her back, with the shoulders raised and the knees drawn up and supported; and she should be covered by a chemise only, or a single sheet of a soft quality, as stiff linen creaks under the stethoscope. The abdominal walls are thus completely relaxed, so as to allow the fœtus in utero to be readily felt, the situation of its back tolerably well ascertained, and the stethoscope to be pressed down into solid contact with the uterine tumour. This depression of the instrument should be exercised solely with the head of the auscultator, and not with his hand, as the bruit musculaire of the latter greatly obscures the fœtal sound. The impossibility of exercising similar depression when the ear alone is employed, constitutes the advantage of *mediate*, over *immediate* auscultation in the exploration of the abdomen. Profound silence is desirable; the auscultator should hold his breath, and he will find his delicacy of hearing increased by also opening his mouth. He should carefully avoid a stooping position and the slightest bend of the neck, both of which circumstances impair the hearing by causing congestion of the head. These two latter reasons constitute almost as strong objections to short stethoscopes as to the naked ear.

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* Dublin Med. Transac.

† Dublin Hosp. Reports, vol. v.

‡ On the application of auscultation to the practice of midwifery, in the Archives Gén. de Med., tom. xxviii. Paris.

The abdominal sounds from which, or through which the auscultator has to distinguish the fœtal beat, are, *a*. The sounds of the mother's heart, sometimes audible on the abdomen, which may easily be discriminated by their synchronism with her pulse and anachronism with the fœtal beats. *b*. Intestinal borborigmus. The listener must wait till it is over. *c*. ***Bruit musculaire*** of the abdominal parietes. The pressure of the stethoscope scarcely excites it if the parietes be well relaxed by position. *d*. Loud arterial and venous murmurs, to be described under the next head. They occur principally in anæmic subjects. It is sometimes difficult for a novice, on a first trial, to separate, as it were, and identify the fœtal tic-tac amongst so many other sounds: once distinguished, it is so peculiar as never afterwards to be forgotten. We now proceed to consider the fœtal beat itself.

The beat of the fœtal heart has been heard by Dr. Kennedy as early as the end of the fourth month. I have repeatedly heard it at four months and a half, and earlier, provided that the mother's calculations were correct. Velpeau and Bouillaud say four months and a half. Laennec therefore placed the date rather too late in fixing it at the "beginning of the 6th month and sometimes a little earlier." Dr. Montgomery says he has not been able to hear the beat before the completion of the 5th month.

The beat exactly resembles that of a young rabbit or kitten, and is closely represented by the *tic-tac* of a watch thickly covered by a pillow. It is feeble during the first half of the fifth month, but by the end of the month it becomes strong and distinct, when listened to in the best situation, presently to be described. M. Dubois says that when the beats are quite distinct, they are *very frequently* attended with bellows-murmur, and he ascribes this to the mixture of the two columns of blood of the pulmonary artery and the aorta. Bouillaud has heard an approximation to bellows-murmur in a few cases. I do not happen to have noticed it.

The speed of the fœtal beat, I have found to be as high as 160 and sometimes a few beats more, during the 5th month: during the 6th, it falls to 150; and during the 7th, to 140: at the full term I have occasionally, though not often, found it as low as 120. It is liable to sudden accidental accelerations, even during stethoscopic examination, and often without any disturbance in the circulation of the mother (Kergaradec). I have frequently noticed this, and it may be the reason why some have rated the pulse as high as 165 so late as the 7th month. On the contrary, I have sometimes known the fœtal beat suddenly become very slow and languid—mostly during faintness of the mother, but sometimes without assignable cause, for disturbances of the mother's circulation do not always influence that of the fœtus.

The spot where the fœtal beat is most audible, varies according to the position of the infant, which, especially during the 7th and 8th months of pregnancy, is perpetually changing its posture. The part of the uterus to which the rounded back and shoulders of the fœtus are applied, is that where the sound is most audible; since, if the stethoscope be well pressed down, not only any interposed intestine, but also the liquor amnii is displaced, and the back of the infant, the uterus and the abdominal walls form one solid conductor for the sound. This part will most commonly be found on one side or other, in a line from the umbilicus to the anterior inferior spinous process of the ilium, but occasionally it is more centrical. During the early part of the 5th month, the part is very low down, because the uterus has not risen high into the abdomen. Its extent after the 5th month may be considerable—equal to an expanded hand. The sound diminishes in intensity on receding in any direction from the focus of the part in question. On commencing his examination, the practitioner should place his hand on the relaxed abdomen and feel for the hardest and most prominent part of the uterine tumour, where, in the great majority of instances, he will at once find the tic-tac in full perfection. If he fail, he should try the other side: if he again fail, he should incline the mother to the sides as he successively examines them, since the movement may cause the back of the fœtus to gravitate to the dependent side. If he still be disappointed, he should apply a cold hand to the abdomen, or exercise some manipulations, which may occasion a favourable change of position by exciting the efforts of the fœtus itself; for, as Laennec remarks, it may probably be sometimes so placed as not to touch the anterior half of the uterus with any part of its back, and such he imagines to be the case when the sound is not audible for hours or days together (ii. p. 459). By attention to the above rules, however, I have seldom failed to discover it in a few minutes. Once, when showing it to my colleague, Dr. Robert Lee, the search cost me a quarter of an hour, but the tic-tac was finally discovered in great perfection.

So constant and so unequivocal is this sign that I have never found it absent in pregnancy with a living infant, though I may have examined upwards of a hundred cases. When the infant is dead, it is of course absent, and practitioners have occasionally failed to discover it, under special circumstances, even when the fœtus was alive: for instance, in pregnancy complicated with ascites (Dr. Montgomery, Cycloped. of Med. Signs of Preg. p. 478). Its absence, therefore, must not hastily be considered conclusive against the existence of pregnancy. The great advantage of this mode of exploring pregnancy, consists in its delicacy and certainty. The most timid rarely object to it, and, after the middle of the term, if the tic-tac be heard, it not only supersedes all other modes of examination, but enables us to affirm that the fœtus is alive. In Forensic Medicine, the sign, from its certainty, is of course invaluable.

In case of pregnancy with twins, it has been found possible to ascertain this circumstance by the existence of two fœtal beats, at different parts, corresponding neither with each other nor with the maternal pulse. It is also possible, after the delivery of one infant, to ascertain the presence of a second. For further details respecting the application of auscultation to parturition, the reader is referred to Kergaradec, Kennedy, and P. Dubois.

2. *The murmur usually called utero-placental.* Others have denominated it the *uterine murmur*: others, again, the *placental*, according to their respective opinions as to its origin. M. Kergaradec had the honour of its discovery.

This murmur is considered to be audible earlier than the beat of the fœtal heart. Laennec says, "we ordinarily *begin* to hear it towards the 4th month," by which he means the end of the 3rd: he then adds, "From the time that the fundus of the uterus has passed the brim of the pelvis, and can be brought into contact with the abdominal parietes by pressure with the stethoscope, we hear the murmur very distinctly, and perhaps even louder than at the end of pregnancy" (ii. p. 461). This rising of the uterus takes place in the course of the 4th month of gestation. Dr. Montgomery does not believe that the murmur can be heard at an earlier period than this, and he has not personally been able to hear it before the completion of the 4th month. Velpeau thinks that if Laennec and M. de Lens really heard it before the end of the 3rd month, it is impossible, for that reason alone, to ascribe it to the utero-placental circulation (Traité des Accouchemens, i. p. 190-1). Dr. Kennedy states that he has frequently detected it in the 10th, 11th, and 12th weeks. It will presently be seen that these discrepant statements are reconcilable on the grounds that there are several distinct sources of the murmur.

It is exactly synchronous with the maternal pulse. In some cases, it exactly resembles that produced by compressing any considerable artery, being the ordinary arterial whiff (Bouillaud). In others, and these are the great majority, it has a prolongation running into the next arterial pulsation; in other terms, it is a *continuous* murmur with augmentations of intensity at each arterial pulsation (Dr. Kennedy). I have also heard it *continuous with little or no augmentations.*

Laennec remarks that, in the later stages, the bellows-murmur is almost always *dull and very diffused*, no longer conveying the impression of being confined to a single artery. All are agreed that it is sometimes slightly whistling (Laennec, Kennedy, Forbes, Bouillaud, &c.).

The iliac regions are the situations in which all concur in thinking that the murmur is most frequently found; yet it is stated that there is no particular part of the uterine tumour where it may not exist (Dr. Forbes, Cyclop. Auscult. p. 242). It is most frequently limited, says Laennec, to a space of three or four square inches, but it may sometimes be heard over an extent exceeding that of an expanded hand: as a general rule, however, it is more local and limited than the beat of the fœtal heart. According to Laennec, Kennedy, Forbes and Montgomery, the spot where it exists does not alter during the course of the same pregnancy, but it may vary in different pregnancies and in different individuals. The murmur, according to Laennec, is not constant. "There are days," says he, "when we can scarcely find it, and we often hear it cease and recommence under the stethoscope without moving the instrument." Kennedy also says, "Intermissions will occur in this phenomenon, upon what cause depending I cannot say."

Respecting the origin and seat of the murmur, I transcribe the following summary from Dr. Forbes (Cycloped. i. p. 242), as it represents the commonly received opinion. "There can be no doubt that the murmur has its seat *in the enlarged vessels of the uterus, in that portion of it immediately connected with the placenta*. This is proved by the following facts:—1. The sound is confined to a fixed space in each individual. 2. This spot is ascertained, by examination after delivery, to be always that to which the placenta had been attached. 3. That the sound is not seated, at least exclusively, in the placenta, is proved by the fact that the sound is still audible for a short period after the placenta is detached. 4. It ceases immediately upon the contraction of the utero-placental arteries, as is proved in cases of death of the fœtus without delivery, and by its instantaneous cessation on the contraction of the uterus after delivery. 5. It is in all cases synchronous with the mother's pulse."

I am disposed to believe, for reasons which will presently be submitted, that the whole of these conclusions are incorrect, and that the entire subject has hitherto been imperfectly investigated.

I believe that Kergaradec, Kennedy, P. Dubois, &c. are in error in restricting the murmur to the uterus, the placenta, or to both; that Bouillaud is equally in error in restricting it to the great arteries of the pelvis; and that all are in error in restricting it to the arteries to the exclusion of the veins.

I venture to submit for further investigation the following propositions:—

1. That the murmur is arterial when it is a whiff.

2. That it is venous when continuous without augmentations synchronous with the pulse.

3. That it is arterial and venous conjoined when it is continuous with augmentations.

4. That its seat is sometimes in the vessels of the abdominal parietes, as the epigastric, circumflexed ilii, internal mammary, and their branches and concomitant veins; sometimes in the great arteries and veins within the cavity of the abdomen, as the common and external iliacs, the renal, the three branches of the cœliac, the colica dextra, media, sinistra and ileo-colica, and the portal veins; sometimes, possibly, in the uterine walls, and sometimes, possibly, in the vessels of various tumours.

5. That the murmur is generally created by pressure, whether that of the uterine or other tumour or of the stethoscope; and that it does not exist independent of pressure except, possibly, in anæmic cases.

6. That the stretched condition of the arteries, and especially the veins of the abdomen, is favourable to the operation of pressure in producing the murmur.

These propositions cannot be adequately comprehended except by one who is thoroughly imbued, both theoretically and practically, with the doctrines of venous murmur developed in a previous section (p. 110). Referring the reader to that section, I shall first show the analogy between abdominal murmurs and those observed in the neck, both arterial and venous, and shall afterwards point out the unsubstantial nature of the arguments which would restrict the murmur in question to the uterus and placenta.

The description of the reputed utero-placental murmur which I have offered above (p. 131-2), is designedly drawn from the ablest writers on the subject, for the purpose of showing that even their own account is identical with that which applies to ordinary anæmic murmurs in the neck. The murmur is sometimes a mere arterial whiff: so it is in the carotids. Sometimes it is continuous with augmentations: so it is in the carotids and internal jugulars, &c. Sometimes it is continuous with little or no augmentations: so it is in the jugulars. Sometimes it is whistling: so it is in the jugulars. Sometimes it ceases without assignable cause: so it does in the jugulars to those who do not understand its nature and the mode of exciting and suspending it. It is most marked in anæmic subjects: so it is in the neck.

A few instances of the several varieties of murmur, as occurring in the abdomen, will now be offered, for the purpose of substantiating the above statements.

1. Respecting the arterial whiff.

Case 1. I lately attended a gentleman, in consultation with Drs. Abercrombie, Monro and Chisholm, in whom an aneurism of the abdominal aorta presented a whiff synchronous with the pulse, which was supposed to proceed from the aneurism itself, but which I believed to be superficial from its high key (the whispered *r* sound), from its being restricted to a line crossing down the summit of the tumour, and from its ceasing whenever the stethoscope was pressed firmly down, so as to obliterate the artery. It was ascertained, after death, to have proceeded from the superior mesenteric artery, which crossed the top of the tumour.

This case is introduced for the purpose of evincing that an artery, even though not of very considerable magnitude, may occasion a high-keyed, *near-sounding* whiff through the thickness of the abdominal walls. *A fortiori*, an artery seated within the substance of the walls, would, *cæteris paribus*, do the same. The case also shows that a tumour beneath an artery, partly by affording counter-pressure, and partly, perhaps, by placing the vessel on the stretch, is favourable to the production of a murmur.

The next cases are instances of the arterial whiff in the large arteries within the abdomen, exclusive of the aorta; for I deem it superfluous to adduce instances of the whiff in this vessel, as the phenomenon is of daily occurrence in anæmic subjects examined in the horizontal position.

Case 2. An emaciated, anæmic, phthisical man at St. George's

Hospital, November 29, 1838. P. 90, weak. The beat of a deep-seated artery could be felt on each side of the umbilical region, crossing from the aorta; (the renal arteries or the colica dextra and sinistra?) and on pressing the stethoscope down upon them, a slight whiff synchronous with the pulse was immediately heard, but without continuous murmur. The same was heard along the common and external iliacs on each side.

Case 3. Another man, examined at the same time, with similar symptoms, presented the same phenomena.

The next case shows that an artery nearer the anterior walls of the abdomen yields a much louder and nearer-sounding whiff, and that a degree of venous murmur is produced in the accompanying vein.

Case 4. James Franklin, in St. George's Hospital, November 29, 1838, had pulsation along the whole aorta, but much stronger and more superficial in the epigastrium than elsewhere. Aneurism of the cœliac artery had been suspected. An arterial whiff was audible all up the aorta; but, at the upper and left edge of the epigastrium, there was an exceedingly loud, superficial and *prolonged*, though not quite continuous murmur. It appeared to run in the line of the splenic artery and vein. I infer that the murmur was partly venous because an artery alone never yields a prolonged murmur. A tumour, whether aneurismal or otherwise, subjacent to the splenic vessels and thus stretching and compressing them, would account for this murmur.

The cases hitherto adduced have illustrated the arterial whiff more especially. We now proceed to—

2. Continuous murmur with augmentations.

Case 5. A female at St. George's Hospital, November 29, 1838, æt. 25, single, of full habit, but extremely pallid and anæmic; P. 100; abdomen tumid from flatulence. Heard continuous murmur with augmentations corresponding to the pulse, on each side of the umbilical region, far from the aorta. The continuous murmur sometimes ceased and left the arterial whiff alone. This occasionally happened for a few moments at a time, though the stethoscope was not moved from the ear, and the cessation was always connected with an audible movement of flatus under the end of the instrument. The murmur only existed in a limited tract.

This case satisfactorily proves that the continuous murmur with augmentations is compounded, in the abdomen as well as in the neck, of the arterial whiff superadded to the continuous venous murmur. It likewise shows that here, as in the neck, the venous murmur may be suspended by slighter causes than suffice to stop the arterial whiff.

Case 6. A girl, æt. 19, in St. George's Hospital, February 6, 1839; very pallid and anæmic, P. 110, with loud venous murmur on the neck. Placed her on her left side, and examined the right. About a hand's breath in front of the anterior, superior spinous process of the ilium and two fingers' breadth above it, a murmur was heard under the following circumstances. On firmly pressing down the stethoscope, nothing was heard; but, after five or ten seconds, on easing the pressure, a sudden, loud venous rush was heard, exactly as when the finger is raised from the internal jugular vein. The rush ceased after four or five seconds, as if the gorged vein had unloaded itself. It was reproduced ten or twelve times by the same process, but it was always weaker, and sometimes failed, during inspiration, as if this act displaced the vessel from beneath the stethoscope. I apprehend that the rush was occasioned by congestion of the vein resulting from its previous obliteration by pressure; for I have shown this to be the explanation of the same phenomenon in the neck (p. 114). I now eased the pressure in a less degree than before, and thus converted the rush into a continuous murmur with slight arterial augmentations, and with an intermixture of *delicate musical notes of humming and whistling.* The sounds continued, as before, to diminish during inspiration. On moving the stethoscope from a particular spot, the murmur was lost and not easily found again.

This case not only exemplifies the venous murmur dissociated, as it were, from the arterial whiff, but shows that the murmur may be musical.

I feel a difficulty in positively determining what vessels were the seats of the murmur in the two preceding cases. The internal epigastric is too centrical. The external epigastric and the branches of the circumflexa ilii are probably too small. The renal are too deeply seated and are higher up; and I have great difficulty in fixing upon the common or external iliacs, because I shall presently show that precisely the same murmur occurs in exactly the same spot in the 9th month of pregnancy, when the uterine tumour is so large as to preclude the possibility of reaching the iliac vessels by pressure. Besides, the murmur is far too loud and near-sounding to be so deeply seated. The most probable vessels appear to me to be the colica dextra and sinistra, vessels not much smaller than a goose-quill, and whose accompanying veins are very large. This idea is perhaps countenanced by the effect of intestinal movements from flatulence or inspiration in interrupting the murmur. The point, however, is open for further investigation.

Though the subjects of the two preceding cases were single, the murmur, it must not be forgotten, was perfectly identical in its nature with that which authors describe as accompanying pregnancy. This will be seen in the following cases.

Case 7. A female at St. George's Hospital in the 8th month of pregnancy. By placing her completely on her side and applying the stethoscope in certain parts, (probably the tract of a considerable artery and vein,) but not in others, a murmur may be produced at pleasure *on either side*; and, further, by a gradual, firm depression of the instrument, this murmur may be made to swell (exactly as in the internal jugulars) into a remarkably loud, diffuse, continuous rumbling sound, augmented synchronously with the maternal pulse.

The next case exhibits the circumstances attending the same murmur with greater precision.

Case 8. Examined a woman at the St. Marylebone Infirmary, Feb. 2, 1839. She was pale, anæmic, excitable, with a pulse of 110, and in the middle of the 9th month of pregnancy. Over the extent of a hand's breath on each side of the uterine tumour, I could hear a very obscure and distant-sounding murmur, on a key below a whispered *who*. It corresponded with the pulse, and, though rather prolonged, was not continuous. I now turned the patient completely over on her right side, so as to cause the uterine tumour to gravitate away from the left os ilii, and leave a soft space as broad as a hand between it and the anterior superior spinous process. I passed the stethoscope gradually along it, taking care to avoid the tumour. On arriving nearly opposite to the anterior superior spinous process, a continuous murmur with augmentations corresponding to the pulse became perceptible, and, with moderate pressure, gradually swelled to so great a degree of loudness that the midwife, though a stranger to the stethoscope, could readily distinguish it. The key was that of a whispered *awe*, it sounded close to the ear, and *a slight degree of whistling was mixed up with it.* On placing the stethoscope on either side of a certain line, the murmur became inaudible, but it recurred as often as I replaced the instrument upon the line itself. Moderate pressure elicited the loudest murmur. Strong pressure diminished it and rendered it less decidedly continuous,—in consequence, I presume, of nearly obliterating the vein. I now made the patient change sides, and I found exactly the same phenomena on the opposite side, but rather less marked.

What was the seat of this loud continuous murmur? Certainly not the uterine tumour, *as I carefully explored far from it.* Unquestionably the murmur was referable either to vessels of the abdominal walls, (branches of the circumflexa ilii artery and vein?) or, what is more probable, to the colica sinistra, a branch of the inferior mesenteric, and its veins,—the latter yielding the continuous murmur, in consequence of being stretched by the weight of the uterine tumour, and of being charged with attenuated blood, and the artery supplying the usual augmentations. What was the seat of the obscure, remote, low-toned murmur audible on the uterine tumour itself? Probably, deep-seated veins and arteries, as the common and external iliacs, compressed by the tumour. I suspect this because, if it had been created in the uterine walls themselves by the act of pressure, it would, I think, have been on a higher key and more near-sounding. At the same time, it is open to investigation whether a murmur may not be excited in the large veins and arteries of the uterine walls by mere pressure. I have not at present cases to decide this point.

I subsequently examined three healthy patients with pulses of 60 to 70, and in the 8th month of pregnancy. In one, I could barely distinguish the remote murmur on both sides of the tumour, and in the other two I could distinguish no murmur whatever. Thus, we are not to expect the abdominal venous murmur in all cases, and its absence will, I think, generally be found connected with the thick, fibrinous blood of good general health, and with the slow pulse that usually accompanies it.

Case 9. A female at St. George's, Feb. 4, 1839, in the 5th month of pregnancy, pale and anæmic from late uterine hæmorrhage. She was examined in the same way as Case 8, and presented exactly the same phenomena, but the murmur was not quite so loud. Both in this and the other case, I tried every other part of the abdomen, but nowhere else than at the points described on either side, could I detect the loud continuous murmur.

3. The continuous murmur with little or no augmentations.

Case 7 was an instance of this. I think I have repeatedly heard it, but I cannot, at the moment, adduce other cases on which I can depend.

Other tumours besides the gravid uterus may produce the reputed utero-placental murmur under consideration. On this point, I shall gladly adduce the evidence of other authors, as they happen to have supplied it.

According to Dr. Montgomery, abdominal tumours of any kind may produce a murmur so exactly like the uterine souffle, that "the nicest and most practised ear cannot detect any difference." He quotes a case lately under his care, "in which enormous enlargement of the uterus, of that kind which has been called vascular sarcoma, was accompanied by this phenomenon in its most perfect condition; and in another case of abdominal tumour, (supposed to be of the spleen,) pressing on the aorta, this sound was equally distinct: moreover, it may at any time be imitated by pressing the end of the stethoscope over the region of the iliac vessels" (Cycloped. Pregnancy, p. 484). M. Bouillaud, again, relates two cases, in one of which "an ovarian tumour *coincided* with a *bruit de soufflet* exactly like that which occurs in pregnancy;" and in the second, a cyst of the right ovary presented a murmur, which was decided by six or seven physicians and a number of students to simulate the placental souffle so exactly that it was actually mistaken for it; the disease was pronounced to be an extra-uterine pregnancy, a vaginal cesarian operation was performed, and the patient died (Traité, i. p. 243).

These writers do not specify the particular kinds of murmur which they heard, whether arterial, venous, or both; but it is probable that they heard the several varieties in different cases.

The cases now offered are perhaps sufficient to establish the fact that the reputed utero-placental murmur may exist wholly independent of pregnancy.

The arguments are now to be examined which have for their object to show that the murmur in question is necessarily seated in the uterus.

Laennec, following Kergaradec, employs both negative and positive reasons for maintaining this doctrine. He argues negatively by excluding other sources of the murmur. The following are his words. The "hypogastric (internal iliac) and primitive iliac" arteries are not the seat of the murmur, "because," says he, "if such were the case, it would exist on the two sides of the uterus at once, or sometimes on one side and sometimes on the other, in the same individual; we could even occasion its production on the one side or the other by varying the position of the subject, and throwing the pressure sometimes on the artery of the left side and sometimes on that of the right—*the whole of which is not the case.*" The preceding cases prove to demonstration that this illustrious observer was in error. He argues positively, by adducing a statement of M. Ollivry; viz. that the murmur ceases "*at the very instant that we cut the umbilical cord*,"—which fact Laennec pronounces to be entirely decisive. It would undoubtedly be so, were it true; but it has been contradicted by Dr. Kennedy, an authority not inferior to M. Ollivry. "Neither does the sound, says he, invariably cease, (as we might be led to conclude from Laennec's statement on the authority of Dr. Ollivry,) on the separation and expulsion of the placenta; but, provided the uterine arteries at this part, *from imperfect contraction of the uterus*, continue pervious to blood, a souffle will still remain, *abrupt however, of short continuance, and wanting the lengthened terminating whiz, observed in the perfect placental sound*" (Dub. Hosp. Rep. v. p. 244, 1830). In short, the venous portion of the murmur ceases, and leaves the mere arterial whiff. May not this change be the result of an alteration in the volume of the uterine tumour, which is diminished, indeed, by the expulsion of the fœtus and placenta, but, in consequence of the *imperfect contraction of the uterus*, is not wholly reduced? May not the smaller size of the tumour suffice to produce the arterial whiff in vessels exterior to the uterus, though it is insufficient to produce the venous murmur? I submit these questions with diffidence; as it is only by one who is expert both as an auscultator and an accoucheur, and who is also thoroughly and dispassionately conversant with the phenomena of the venous murmur, that they can be solved. If Dr. Kennedy could find leisure to revise the subject, he would confer an additional benefit on obstetric science.

Dr. Forbes states, in his Conclusion 4, that "the murmur ceases immediately on the contraction of the utero-placental arteries, *as is proved in cases of death of the fœtus without delivery.*" This, however, is also contradicted by Dr. Kennedy, who says that it does not always *cease* on the death of the child, but "*has its character altered from the* CONTINUOUS *murmur with its lengthy, sibilous termination*, to an abrupt, defined, and much shorter sound." This is the arterial whiff as before, and apparently created by the same circumstance—the diminished volume of the uterine tumour, connected with the death of the child and commonly with the rupture of the membranes.

To afford direct demonstrative proof that murmur does not exist in the uterine arteries supplying the placenta, is not easy. Such proofs would best be collected by auscultators who are also accoucheurs, and to such I would beg to recommend the subject. Meanwhile, it may be remarked that the view which localizes the murmur of pregnancy in the utero-placental arteries, is not very reconcilable with general analogy. Assuming, for a moment, that the uterus is one of the erectile tissues, why is not its murmur constant, as it is in the case of real erectile tumours,—such, for instance, as varicose aneurism? If it be replied, with Laennec, that the interposition of intestine might occasionally prevent it from being heard for a few minutes, hours, or whole days, as Laennec states, I would venture to rejoin that this explanation is unsatisfactory; for any one may satisfy himself, by finding dulness on percussion, that intestine positively is not interposed over a great portion of the anterior and lateral parts of the fundus of the uterus. If, therefore, a stethoscope were applied on any of these dull portions, a murmur generated in the utero-placental arteries would infallibly be heard through the solid medium of the abdominal and uterine walls. It must be recollected, in exemplification, that a murmur of the heart or ascending aorta can often be heard through the whole thickness of the dorsal and even scapular muscles.

But if the uterus be not an erectile tissue, murmur in its arteries ought to be amenable to the same general laws as regulate its production in arteries in general. Now these laws point out that arterial murmurs, when not created by local pressure, are restricted almost entirely to thin-blooded subjects and to periods of vascular excitement. But the utero-placental theory requires that the murmur should exist in all subjects and on all occasions indiscriminately, and it does not assume that the murmur is created by local pressure. Here then is an inconsistency.

Such are the reasons which lead me to believe that something like the propositions with which I commenced this article (p. 133), will ultimately be established as the doctrines relative to abdominal murmurs connected with the gravid uterus, tumours, &c. I do not pretend to have offered more than a general sketch or outline of the subject, which the investigations of others will probably fill up and correct.

Meanwhile, the immediate practical conclusions are as follows.

1. A near-sounding, high-toned continuous or venous murmur with arterial augmentations, heard opposite to the anterior, superior spinous process of the ilium and a little above, does not necessarily indicate pregnancy, because it may exist in connexion with other tumours, and also wholly independent of any tumour. It occurs almost exclusively in the thin-blooded or anæmic with a quick pulse.

2. An obscure, distant, low-toned murmur, synchronous with the pulse and not continuous, though sometimes rather prolonged, heard on a tumour in the hypogastric region, affords presumptions that the tumour compresses the iliac vessels.

3. When either or both of the murmurs coincide with other symptoms of pregnancy, they afford presumptions of this state, but do not warrant an affirmation.

PART II.

INFLAMMATORY AFFECTIONS OF THE HEART AND GREAT VESSELS.

THIS Part will be divided into four chapters. The first will be devoted to inflammation of the external membrane (pericarditis); the second, to that of the muscular substance (carditis); the third, to that of the internal membrane (endocarditis); and the fourth, to that of the internal membrane of arteries (arteritis). The close connexion subsisting between inflammation of the external and internal membranes of the heart,—in other terms, the frequent coexistence of pericarditis and endocarditis, will be pointed out in the chapter on Pericarditis.

CHAPTER I.

ON PERICARDITIS.

SECTION I.

ANATOMICAL CHARACTERS OF PERICARDITIS.

The anatomical characters of acute inflammation of the pericardium are, 1. preternatural redness of the membrane; 2. coagulable lymph adhering to its surface; and, 3. fluid effused within its cavity. They will be treated of in succession, and at some length. For, as the anatomical characters are an important key to the symptoms, the latter cannot be understood, and, consequently, so rapid and fatal a malady cannot be treated with the promptitude and decision essential to the safety of the patient, unless the characters in question, and their intimate connexion with the symptoms, are thoroughly known to the practitioner. To this subject, therefore, I would particularly direct the attention of the student.

1. *Preternatural redness of the Pericardium.*—The redness is seated partly in the serous membrane, but still more in the subjacent cellular tissue. It very seldom pervades the whole of the inflamed portion. It presents itself sometimes in numerous small scarlet specks with a natural colour of the intervening membrane, sometimes in spots of greater or less magnitude formed by the agglomeration of the specks, sometimes in the form of arborescent and stellated vascular injection, and

sometimes in patches or diffuse redness of considerable extent, formed by coalition of the spots, or thickening of the arborescent injection. Both the patches and diffuse redness, however, have, almost without exception, a dotted or mottled character. In a drawing before me, which I made from a case of very acute and rapid pericarditis, nearly the whole of the reflected membrane, underneath a layer of soft, primrose-coloured lymph, is of a vivid, diffused, but mottled and dotted red (See the writer's Morbid Anatomy, Fig. 54). In some cases, according to Laennec, though the inflammation, judging of it by the thickness of the false membrane, had been very severe, scarcely any redness exists. Such is the case, with respect to the surface of the heart, in the drawing to which I refer. Here, it is to be presumed that the redness existed during life, but vanished after death, as it is wont to do in arachnitis, pleuritis, ophthalmia, and many other diseases, when the inflammation is either very recent or only slight—when, in other words, the blood has not yet become stagnant and impacted in vessels. This is important to be known, lest the absence of redness should lead us to deny the existence of pericarditis, or to imagine that the inflammation was confined to the few spots to which lymph happens to adhere,—a mistake which I have often seen committed.

When acute pericarditis degenerates into chronic, the redness loses its brilliancy, sometimes becoming very deep and of a brownish colour, and sometimes acquiring a cinnamon hue.

Redness *alone* does not afford conclusive evidence of pericarditis, as all serous as well as mucous membranes are liable to vascular injection from various causes independent of inflammation; especially, obstruction to the return of the venous blood by valvular disease, dilatation, softening, &c.; the diseased, incoagulable state of the blood in typhus, scurvy, purpura, &c.; and imbibition of blood effused into the pericardium in hæmorrhagic pericarditis, of which I have seen several instances (See the writer's Morbid Anatomy, Fig. 61). In all these cases, indeed, the redness has a more uniform intensity and a more abrupt outline,—in short, it is more like a stain, than inflammatory redness. Still, as it is very possible to mistake one for the other, redness should not be considered as affording conclusive evidence of pericarditis, unless conjoined with an effusion of lymph or sero-purulent fluid.

As the sub-serous cellular tissue is softened by acute pericarditis, the serous membrane admits of being peeled off with preternatural facility.

The pericardium, according to my observation, very rarely undergoes thickening, and then, only in a slight degree, that which is often regarded as thickening being, in general, nothing more than superimposed and intimately adherent false membrane of old standing, and of opake, bluish-white appearance. I have seen this appearance pervade the whole surface of the heart under a layer of old, cinnamon-coloured lymph; yet, after peeling off the lymph, I could, by further scraping, remove the white layer also, without injuring the surface of the pericardium (See ibid. Fig. 64.)

2. *Coagulable lymph adhering to the surface of the pericardium.*—The inflamed pericardium secretes serum and lymph conjointly, and in a fluid state, from the same vessels. The process may commence almost simultaneously with the inflammation. The absolute and relative quantities of the serum and lymph vary greatly in different cases. I have known serum secreted in such quantity and with such rapidity as to amount to a pint in twenty-four hours: on the other hand, I have frequently found it so scanty, especially in the early stage, as not to separate the surfaces of the pericardium and prevent the sound of attrition,—a sound which they yield, as will be hereafter explained, when roughened by adherent lymph. Soon after the secretion has taken place, the lymph separates from the serum by concretion, and adheres to the membrane. A small proportion, however, generally remains suspended in the serum in the form of flakes and filaments. The adherent lymph, when recent, is of a pale straw colour, and of a soft, tender consistence, becoming firmer and more tenacious as it grows older. Though occasionally deposited in detached lumps and spots, the latter imparting to the surface a rough, papulated or granulated character, it generally forms continuous layers, sometimes covering a portion only, but more commonly the whole, or nearly the whole, of the pericardium. The thickness of the deposition may vary from a line to an inch;

but from a line and a half to three lines is its ordinary mean. Its adherent surface is smooth; the opposite is rough and singularly figured. In drawings before me, where I have delineated from nature all the appearances that I have ever witnessed, the free surface is sometimes pitted with small depressions at tolerably regular intervals, presenting the aspect of a fine reticulation or of the section of a sponge. This occurs principally where the layer is thin; where it is thick, the surface is distributed into more spacious cells, often as large as a pea, and separated by coarser partitions. The partitions are sometimes irregular, being higher and thicker in one part than another; in which case the effect exactly resembles that produced by separating two flat plates, between the surfaces of which a layer of soft butter has been spread. At other times the partitions are very regular; in which case, the appearance, as Corvisart observes, is analogous to that of the second stomach of a calf. Occasionally they are very thick and rounded, and then they have an appearance somewhat similar to that of a congeries of small earth-worms. Not unfrequently they are shaggy and flocculent, hanging in shreds like tow. In one drawing, from a case which had become chronic, no cells are apparent, but the lymph is arranged in transverse, and, as it were, plaited wrinkles, like undulations of sand on the sea shore.*

As coagulable lymph on other serous membranes does not present these peculiar arrangements, except occasionally on the pleura, they must be referable to the perpetual movements of the pericardium, or, as M. Bouillaud happily expresses himself, "to the incessant repetition of the experiment above described, namely, the sudden separation of two surfaces overspread with a soft matter, of the consistence of soft butter." The pleura is subject, though in a less degree, to the same friction: hence it is, that it occasionally presents a honeycomb appearance. When lymph becomes old, it acquires a deeper hue, varying from cinnamon to an intense brown-red or mahogany colour. When of the latter colour, it usually secretes bloody fluid, and, as well remarked by Laennec, it is to the stain of this blood that the dark-red colour is attributable. He has denominated such cases "*hæmorrhagic pericarditis* (Traité, ii. 654).

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* The whole of these appearances are delineated in the writer's Morbid Anatomy, Figs. 54 to 64.

The organization of lymph sometimes takes place with astonishing rapidity, as within the space of twenty-four hours—a fact which has been ascertained by experiments on living animals, and by pathological observation on the human species. The exceptions I have generally found to occur either in very intense inflammation, when the violence of nature's operations would seem to counteract their sanatory tendencies, or in atonic and cachectic subjects of bad constitution, in whom the lymph effused is of an unhealthy character and ill suited for organization, just as we observe in the case of external wounds affecting the same subjects.

Before describing the process of organization and adhesion, let us pause a moment to ask what is the object which nature proposed to herself in the effusion of lymph. Unquestionably to effect reparation:— the object for which the effusion is designed, in whatever part of the system it takes place. But how, it may be inquired, can it effect reparation in the pericardium? By causing adhesion. Supposing that the inflammatory process does not terminate by resolution—by the complete absorption of both lymph and serum, the most desirable termination which remains is adhesion; for, should this not take place, the lymph becomes a secreting surface, which effuses more and more lymph and serum, until, in a short time, the cavity is completely distended, and the action of the heart so embarrassed that a fatal termination speedily ensues. But, should adhesion of the opposite surfaces take place, by which further effusion is prevented, life may be prolonged for a considerable period--even for years; though, as will presently be explained, the adhesion, so far from being a *perfect* reparation, gives rise to another form of organic disease, which, in a vast proportion of cases, ultimately proves destructive to the patient.

Adhesion takes place in some cases and not in others,—a circumstance which has been attributed to a difference in the quality of the lymph, dependent on the greater or less energy of the inflammation, or on the more or less healthy constitution of the patient, in consequence of which it possesses different degrees of aptitude for organization and adhesion. This explanation, though not unsound, is less applicable to the pericardium than to other serous cavities; for, here, the union or non-union depends also on the absence or presence of fluid in the cavity; the best lymph, equally with the worst, being incapable of uniting when interposed fluid prevents the apposition of the opposite surface. Hence it is that a considerable extent of the pericardium often adheres, while some portion, in which a little remains of fluid had accumulated, does not; and this spot I have of late years observed to be most frequently situated at the angle formed between the base of the heart and the origin of the great vessels: for the same reason it is, that, when the whole of the peritoneum is covered with lymph, the intestines adhere to each other, but their adhesion with the walls of the abdomen is prevented by the interposition of fluid.

Hence the immense importance, in pericarditis, of prompt and energetic treatment in the first instance, in order, if resolution cannot be effected, to cause absorption of the fluid, and thus afford the opportunity for adhesion. Temporizing indecision is inadmissible; for unless one or other of these terminations be induced, the patient inevitably dies.

Such is the object of adhesion: we have now to describe the process. It has already been stated that the organization of the lymph may commence within twenty-four hours. When the fluid has been sufficiently absorbed, the layers of lymph on the opposite surfaces of the pericardium come into contact, blend, and gradually become united by vessels presenting themselves under the successive appearances of blood-stains, straggling lines, and, lastly, of uniform pinkish vascularity, susceptible of injection from the pericardium. The pinkness gradually diminishes, and with its disappearance the organization may be considered complete. The depositions are thus converted into perfect cellular tissue, by which the contiguous parts are more or less firmly, closely, and extensively agglutinated. When adhesion is of recent standing, the lymph is generally thick, and so soft as to be separable by mere tearing into two layers, one adhering to each fold of the pericardium. In proportion as the disease is older, the false membrane is thinner and firmer, consisting, in cases that date several years back, of the finest layer of dense cellular tissue.

In some, even this is not perceptible, the folds of the pericardium having become amalgamated—apparently without the intervention of any membrane, so as with difficulty to be separable, even by the scalpel. (Case of May.) It is in such cases that pathologists have sometimes erroneously supposed the heart to be destitute of a pericardium.

Such is the ordinary progress of adhesion; but in some rather protracted cases, generally of at least two or three months duration, where, though adhesion has been established, inflammation has either recurred or never been completely subdued, an additional interstitial deposition of lymph takes place, which has been known to thicken the adventitious mass to the extent of an inch and upwards. In this case it sometimes possesses a laminated texture, the layers of which are progressively redder in proportion as they are nearer the heart; and sometimes it exhibits different degrees of consistence in different parts, one being almost liquid and purulent, while another has the density of tubercular induration.* Such cases are ordinarily fatal at no very remote period.

Adhesion is not always universal; for, sometimes, though the inflammation has pervaded the whole membrane, the depositions of adherent lymph are only partial: sometimes, again, the inflammation itself is only partial. In both these cases, the adhesions are confined to the portions on which lymph was effused; and when these portions are limited, the adhesions are not close or intimate; for, as the gliding motion of the heart within the pericardium is not prevented, it stretches the adherent lymph, and converts it into long, loose bands of cellular tissue. But when the portions overspread with lymph are extensive, partial adhesions are sometimes close and firm, and the intervening parts of the pericardium may be healthy and in contact. Instances occasionally occur of adhesions being partial, though the layers of lymph are universal; but here the parts not united are separated by purulent fluid, thus constituting a series of small, detached abscesses around the heart. Sometimes lymph is deposited in the form of small, roundish, soft granulations, with which the pericardium is more or less extensively studded.

Laennec is of opinion that pericarditis may sometimes be partial, and confined even to a very limited portion of the membrane; but he adds that such cases are rare, scarcely amounting to one in ten, unless white spots, presently to be noticed, be admitted as falling under the head of partial pericarditis, which will greatly augment the proportion. Partial pericarditis, he pursues, almost always terminates in recovery and transformation of the false membrane into long serous bands (De l'Auscult. tom. ii. p. 655). Without denying that pericarditis can be partial, I doubt whether this can be proved; since the signs, both general and physical, do not differ, except in degree, from those of universal pericarditis, and since adhesions may be partial though the inflammation have been universal, provided the layers of lymph happen to have been only partially deposited. I lately, for instance, examined a heart presenting several detached patches of recent lymph, yet the redness, and probably therefore the inflammation, was universal.

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* Latham Lond. Med. Gaz. vol. iii. p. 5.

Pericarditis sometimes leaves no other vestiges than opake white or milky spots, which are a well-known appearance on the surface of the heart. It is possible that they may be results of partial pericarditis, as supposed by Laennec, but there is no reason to suppose that they may not also be occasioned in some instances by universal pericarditis, as in the case referred to in the preceding paragraph. The spots vary in extent from a few lines to two or three inches in diameter; their thickness is about that of the nail: they commonly consist of a layer of false membrane, which has assumed the character of condensed cellular tissue, and, with a little care, they may generally be detached without injury to the pericardium beneath, which is commonly somewhat injected, though not thickened. But, though this is the ordinary cause of white spots, I have sometimes found them to be occasioned by hypertrophy of the sub-serous cellular tissue, and even of the fibrous layer of the pericardium itself; but I have never seen the serous layer so thickened and opake as to present this appearance.

In concluding this account of the changes undergone by false membrane, it may be added that, in conformity with the laws of embryogony and of the scale of animals, false membrane, like cellular membrane in general, is subject to "*analogous transformations*," that is, a change from the state of cellular tissue to that of fibrous, cartilaginous, and osseous. Hence it is that we occasionally see masses of cartilage and bone, sometimes of surprising magnitude, connected with the exterior of the heart, as described in Chap. VII. on osseous and other productions.

3. *Fluid effused within the cavity of the pericardium.*—It has been stated that serum is effused conjointly with lymph, from the vessels of the inflamed pericardium, and that a separation of the two takes place by the concretion of the lymph. The remaining fluid is occasionally transparent, and either of a faint yellow more or less tinged with green—as that of the interior of a lemon, or of a pale fawn colour; much more commonly, it is somewhat turbid and cloudy from containing flakes, filaments and fragments of concrete lymph, which had not adhered in the form of false membrane, or had been detached from the latter by friction and agitation; occasionally, even in the first stage, it presents some degree of milky opacity from an admixture of real pus; and in a very few cases pure, creamy, greenish-yellow pus is effused without any deposition of false membrane (e. g. Case 4, of M. Bouillaud, vol. i. 336). It will presently be shown that purulent effusion is more common in the chronic stages. The quantity of fluid, though variable and sometimes scanty, is in general considerable at the commencement, that is, within the first two, three or four days of the disease,—not unfrequently amounting to more than a pint. Corvisart once found four, and Louis the same. It is speedily diminished, however, by absorption when the first violence of the inflammation begins to subside; and, after the lapse of a few days, it is, in the majority of cases, not more abundant than the concomitant exudation of lymph. Sometimes, indeed, —even in very acute inflammation, the absorption is so complete that no serum whatever is found, while a copious exudation of thick, concrete lymph fills and agglutinates the whole cavity. Laennec, observing this, asks with his usual acuteness whether lymph may not sometimes be effused in the dry state—a surmise which has since been established as a fact by the occurrence of the sound of attrition of lymph in the first stage of the diseases.

Should complete absorption of both the fluid and lymph not take place, nor yet adhesion of the pericardium be established, but the disease run on in the chronic form, the fluid, if previously only serous or sero-flocculent, gradually becomes more milky and opake, until it eventually assumes a perfectly sero-purulent character. This results from the tendency exhibited by inflammations in general to secrete pus in their chronic stages. Rarely, however, is perfect pus found in the pericardium;—probably because the patient dies from irritation before the suppurative process is fully established. Not unfrequently the fluid is bloody (Cases of Porter and Snowden), and the lymph of a red colour (Case of Porter). This is attributable to the tenderness of all newly organized structures, in consequence of which they are apt to become congested and to effuse blood when subjected to any unusual irritation or excitement, a phenomenon witnessed daily in external ulcers.

Compression exercised by fluid sometimes reduces the volume of the heart, and renders it, as it were, atrophous. Bouillaud relates cases in which the same was effected by "enormous masses of false membrane" (Traité, i. 448).

Such are the anatomical characters of acute pericarditis, both in its early and its advanced or chronic stages. It remains for me to make a few remarks on that form of pericarditis which appears, from the mildness of the inflammatory symptoms, to have been chronic from the first. Its anatomical characters do not differ very materially from those exhibited by the advanced stages of the acute form. The inflammation always pervades the whole of the cavity; the redness is deeper and duller than in the acute affection ; false membranes are, in many cases, totally deficient; and when present, they are thin, soft and fragile, as if wasted by suppuration : finally, there is always a more or less abundant effusion of turbid, flaky, milky, and sometimes completely puriform fluid. *Intimate* adhesions of the pericardium to the heart may follow the absorption of this fluid; but M. Laennec does not appear to me to be borne out either by facts or by analogy, when he supposes that chronic pericarditis is the sole cause of intimate adhesion, and that the acute affection only gives rise to loose adhesion by more or less elongated bands. According to my experience, the latter is the more frequent cause of intimate adhesion (Cases of Copas, May, and many others after acute rheumatism).

In scrofulous and phthisical individuals, tubercles are sometimes developed in the false membranes of pericarditis, and, according to Laennec, they may cause the acute to pass into the chronic state, as frequently happens in the case of pleuritic and peritoneal false membranes.

The muscular substance of the heart is sometimes not affected by pericarditis; but sometimes it is rendered redder or paler, browner or yellower, harder or softer and more lacerable, than natural. These changes result from inflammation propagated from the pericardium to the muscular substance, as will be fully shown in the article Softening.

As endocarditis co-exists with pericarditis in the immense majority of cases, the practitioner must not neglect to extend his observations, in cases of pericarditis, to the interior of the heart, where he will generally find tumefaction and constriction of the valves, redness of the lining membrane, coagula, &c., as will be fully explained in the chapter on Endocarditis.

SECTION II.

SIGNS AND DIAGNOSIS OF PERICARDITIS.

THERE is no inflammatory affection of which the diagnosis has been considered more difficult than pericarditis. Corvisart states that very acute pericarditis is often completely concealed, and that, of chronic pericarditis, he "has always found the diagnosis difficult, and sometimes very obscure." Laennec states that he has often, on dissection, discovered the disease in a severe form, when nothing had afforded a suspicion of its existence; and, on the other hand, that he has frequently witnessed all its signs, without finding a vestige of the malady. He adds that he has seen this double mistake committed by the most expert practitioners, and he therefore concludes that pericarditis can only be guessed at (devinée), not detected (reconnue). Dr. Latham mentions two cases of what appeared to be, and was treated as, marked inflammation of the brain; yet this organ was found perfectly sound, and the heart affected with intense pericarditis (Lond. Med. Gaz. vol. iii. p. 209). Andral relates a similar case (Clinique Medical, vol. iii. p. 444). Others have more recently been published by Dr. Macleod in the Med. Gaz. It is proper to keep these difficulties prominently in view, in order that practitioners may be better prepared to contend with them. But it must be added that such cases as those of Latham and Andral are very rare; and that, with the improvements in diagnosis introduced by modern research, the disease may, I feel assured from numerous post-mortem examinations, be nearly always detected. Since I wrote the preceding sentence, nine years ago, new lights have been thrown on certain of the physical signs, which have rendered the diagnosis still more precise and certain.

I shall first enumerate the general signs, and then endeavour to point out the causes of their obscurity, the means of rendering them available, and the diagnosis from other inflammatory affections of the chest. The physical signs will subsequently be considered.

General Signs.[*] They are as follows: acute inflammatory fever; sometimes a pungent, burning, lancinating pain in the region of the heart, shooting to the left scapula, shoulder and upper arm, but rarely descending below the elbow, or even quite to it. The pain is increased by full inspiration, by stretching the left side, by percussion, and especially by pressure between the præcordial ribs, and forcing the epigastrium upwards underneath the left hypochondrium. In other cases, the pain is more or less dull, and does not lancinate: occasionally, it is wholly absent, or is merely an uneasiness.[†]

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[*] By General Signs, I mean all those not furnished by percussion and auscultation, which latter I shall call physical. The more refined subdivisions which some have preferred, are perplexing without answering any object.

[†] M. Bouillaud says, "I have done my utmost to discover some satisfactory reason for this sort of inconstancy in the pain of pericarditis. The results of a comparative examination of a good number of cases are—1. That the most simple pericarditis is precisely that in which pain is wholly absent, or, at least, but slightly felt. 2. That rheumatic pericarditis also is often free from pain, or at least but very slightly painful, if there is no pleurisy. 3. That in this last complication, there is most frequently, though not always, such a pain as I have described, especially when the pleurisy occupies the left side; and that this pain is never more acute, more poignant, in short, more dreadful (*atroce*), than when the pleurisy is seated on the left side of the diaphragmatic pleura" (Traité, i. 453). I have made researches similar to the above, more especially during the last eight years; and I have certainly found that, in the great majority of cases, the pain was either wholly absent, or of a mild, endurable kind; yet I have occasionally seen it more considerable, in the absence of pleuritis,

The next symptoms are, inability of lying on the left side, and sometimes in any position but one, which is most commonly on the back; dry cough; accelerated respiration; palpitation of the heart, the impulse of which is sometimes violent, bounding and regular, though its beats may, at the same time, be unequal in strength; at other times it is feeble, fluttering, intermittent, irregular and unequal; pulse always frequent, and generally, at the onset, full, hard, and abrupt, or even jerking, but regular. Sometimes it maintains these characters throughout, but more commonly it becomes, after a few days, weaker than accords with the strength of the heart's action, and, in the worst cases, small, feeble, intermittent, irregular and unequal, in accordance with similar action of the heart. Occasionally it possesses the latter characters from the commencement; whenever they exist, they are accompanied by dyspnœa; a constrained position, deviation from which induces a feeling of suffocation; extreme anxiety both of countenance and mind; a peculiar drawn or contracted appearance of the features, occasionally with the sardonic grin; faintness, paleness, failure of animal heat, constant jactitation, insupportable distress and alarm, cold perspiration, and, finally, from obstruction of the circulation, intumescence and lividity of the face and extremities. I have seen extensive œdema of the feet supervene during the last twelve hours of life. Delirium and convulsions are also occasionally seen in the last stage, being results of cerebral congestion and of the circulation of venous blood.

Such is the category of symptoms of pericarditis. One cause of their obscurity would, at first sight, appear to consist, and by Corvisart, Laennec, Louis, and pathologists in general has actually been found to consist, in their diversified, incongruous, and variable nature. The pulse, for instance, displays, at one time or

than M. Bouillaud seems to intimate. I therefore think that further observation will be requisite before we can come to the conclusion that a pleuritic complication is the sole cause of *considerable* pain in pericarditis, though it may be of the dreadful (atroce) pain which he describes, and of which he has given two or three instances—one, in the distinguished orator Mirabeau. M. Andral observed "acute, dreadful, rending pain" (vive, atroce, dechirante) in one case (Clin. Med. iii. 416), and M. Louis found "acute pain" (douleur vive) in two cases (Mem. on Pericarditis); yet in all these instances there was no pleurisy. I have not searched for other cases, but Louis estimates that there is pain in one half.

other, almost every kind of character; the disease, though the inflammation be equally intense, is sometimes very supportable,—at others, agonizing: in one case it terminates fatally in two or three days,—in another it lasts as many weeks!

Now, in reality, these diversities, while they do not render the symptoms less pathognomonic of the disease in general, as will presently be shown, are, according to my observation, invaluable indications in another point of view—they contribute to denote the nature and progress of the anatomical changes of structure, and, in correspondence, the progress and exact state of the malady. For it is a fact of which I feel well assured from long observation, that a difference in the quality and quantity of the effusion imparts a totally different aspect to the symptoms. Thus, when, either from the effusion consisting principally of coagulable lymph, or from the simultaneously secreted serum being rapidly absorbed, universal adhesion of the pericardium promptly takes place, preventing all further fluid effusion, the action of the heart maintains throughout much the same vigour and regularity as it manifested at the onset of the malady, and the pulse exhibits corresponding characters of strength, hardness and regularity.* Under these circumstances also, the position is less constrained, and less pain is produced by an unfavourable one; in consequence, perhaps, of the heart being curbed by the adhesion, and thus prevented from impinging with the same degree of violence against the thoracic walls. Finally, as the force and rhythm of the heart's action, and consequently the circulation and respiration, are adequately maintained, the life of the patient will be prolonged probably for weeks even though the inflammation remain unsubdued, and, if he sink at all from the *immediate* effects of the disease—of which I have never had an instance in my own practice, he will sink apparently from mere exhaustion by the effects of protracted irritation. Now this is a very supportable form of disease, and it is still more supportable if, instead of proceeding to adhesion, it undergo resolution by the effect of remedies,—which is a very common result under the treatment hereafter to be described.

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* Since I wrote the above, Dr. Stokes has recorded four or five fatal cases in which the effusion consisted almost entirely of lymph (Dublin Jour. vol. iv. p. 29); and he supports the doctrine broached in the text.

But the case is very different if, instead of adhesion or resolution taking place, there be a copious serous effusion remaining unabsorbed. The heart's action is then mechanically embarrassed by the compression exercised by the fluid,—a compression which is the more considerable from a double cause: first, because the effusion is sudden, and organs do not so easily accommodate themselves to sudden, as to gradual compression; *secondly*, because the pericardium, deprived of its distensibility by inflammation, is incapable of yielding as the fluid accumulates. Hence the heart, unable to transmit—perhaps even to receive, the blood, flutters, intermits, beats feebly, irregularly, and unequally. The pulse has corresponding characters, and is sometimes scarcely perceptible. From this failure of the circulation through the heart, result its usual symptoms: namely, faintness, dyspnœa, anxiety, coldness, lividity, a sense of suffocation on the slightest deviation from a certain position, with all the other symptoms of an extremely obstructed circulation. If this state be not expeditiously relieved by remedies, the patient dies in the space of a few days or even hours.*

Should the fluid be copious from the first, this series of symptoms will make its appearance equally early; but, in general, two, three, or four days elapse before the accumulation becomes considerable; in which case the former series—those attended with strong and regular action of the heart, will exist during this period, and will then be suddenly replaced by the latter. In a few instances, I have found the latter exist when the quantity of fluid was inconsiderable, but that of lymph enormous. I conceive, therefore, that an enormous accumulation of lymph has the same effect as fluid in embarrassing the action of the heart. I have also found the worst class of symptoms occasioned by a less quantity of fluid in some cases than in others,—a difference which probably depends, in some cases, on diversities in the nervous irritability; but, in others, I suspect that it is connected with the simultaneous existence of carditis; for, when the affection has been thus complicated, I have known the feeble, fluttering action of the heart and all its concomitant train of unfavourable symptoms, occur, though the effusion within the pericardium was inconsiderable; and it is reasonable to suppose that, when the heart is softened by inflammation, its contractile power would be so far impaired as to render it incapable of transmitting its contents. In others, again, the worst class of symptoms may result, according to M. Bouillaud (Traité, i. p. 463, &c.), from polypous concretions in the heart, occasioned by co-existent endocarditis. Though I have never had the opportunity of personally ascertaining this by post-mortem examination, I can easily understand and believe it. The peculiar expression and sardonic contortions of the features attending the worst class of symptoms, are occasioned by the sympathy subsisting between the respiratory nerves of the face and those of the heart; or, if it be not premature to recognise the universal *true spinal* or *excito-motory* system, they are occasioned by the *incident* or *excitory* filaments of the pneumogastric nerve transmitting an *excited* impression to the true spine, whence it is reflected on the face by the *reflex* or *motor* filaments of the portio dura, and portio minor of the Trifacial.*

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* It is instructive to observe that the same class of symptoms is induced under whatever circumstances the circulation through the heart is extremely impeded: thus, I have seen them result from poisoning by arsenic, and from intense gastro-enteritis: they result also from poisoning by the concentrated mineral acids, by tobacco, &c.—all of which agents have a paralyzing effect on the heart. I have likewise seen them occasioned by polypi forming in the heart before death (see Polypus) and by extreme softening of the organ (see Symptoms of Softening).

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* M. Bouillaud explains the great differences in the general symptoms of pericarditis in another way, which to me does not appear satisfactory. Having observed certain cases, (especially Case 5, i. 345,) in which pericarditis *complicated with diaphragmatic pleurisy* presented the most frightful series of general symptoms; and having observed other "cases of very intense pericarditis in which these frightful symptoms scarcely existed at all," he comes rather precipitately to the conclusion that "the severe general symptoms (les grands accidents de reaction) observed in certain cases of pericarditis, ought to be considered as appertaining more directly to a violent pleuritic or pleuro-peripneumonic complication than to the pericarditis itself; since these severe symptoms may be wholly absent in pericarditis exempt from so serious a complication, and, on the other hand, we may see them burst out in exceedingly acute pleurisies and pleuro-peripneumonies, (especially diaphragmatic,) without co-existent pericarditis" (Traité, i. 463). There is no doubt of the fact that pleurisy and pleuro-peripneumony, especially diaphragmatic, may produce the violent symptoms in question: of this, I have myself seen repeated instances: it is therefore obvious that these complications might aggravate the symptoms of pericarditis, and raise them to their utmost intensity; but it does not follow, on this account, that all cases of pure pericarditis should be exempt from severe symptoms. Nor are they: for I have already quoted Andral and Louis' cases (p. 156,) to prove that the pain may be dreadful, and I have shown that when the circulation through the heart is suddenly and extremely impeded, not only by diseases of the organ itself, as compression by

Such are the causes of the general symptoms. It will now be apparent that their variability is calculated to enlighten, rather than to perplex the practitioner, and that, whatever aspect they assume, they would still be abundantly sufficient, did no other difficulties interfere, to render the disease one of easy diagnosis, even without the aid of auscultation. But there *are* other difficulties which render that aid indispensable. These consist partly in the absence or mildness of some of the most important symptoms, and partly in the presence of pulmonary complications. I shall notice them in succession.

When pain in the immediate situation of the heart, increased by pressure in the interspaces between the ribs or upwards under the left hypochondrium, is accompanied by increased action of the organ and fever, there can be little doubt of the existence of pericarditis. But sometimes, and not unfrequently, pain is slight

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much fluid within the pericardium, carditis with softening, and polypus, but also by nervous paralysis of the heart from poisons, intense gastro-enteritic inflammation, &c., the worst symptoms of fainting and apnœa with overwhelming anxiety and "nervous reaction" may be induced. M. Bouillaud, indeed, seems to admit this by implication —never, however, forgetting his favourite pleuritic affection: for he says "the phenomena of dyspnœa carried to suffocation, and those of faintness carried even to syncope, coincide, if not always, at least most frequently, with an immense effusion in the *pleura* and the pericardium, and with the formation of polypous concretions in the cavities of the heart. The extension of the inflammation to the fleshy fibre of the heart and the inflammatory tumefaction of the valves, are also *incidental* circumstances attending pericarditis to which we must attribute an important participation in the reactional phenomena, and particularly in the disturbance of the circulation and respiration" (Traité, i. 462).

M. Bouillaud has here enumerated circumstances enough (and they are those specified in the text) to account for the worst class of general symptoms attending pericarditis, without being driven to the necessity of ascribing them almost entirely to a pleuritic or pleuro-pneumonic complication. I shall, in conclusion, cite a passage from a valuable paper on pericarditis by Dr. Stokes, (Dublin Journal, vol. iv. p. 54, 1834,) who exactly corroborates the view which I have for the last eight years maintained in the text, respecting the principal cause of the diversities in the symptoms of pericarditis. "I shall now give," says he, "the results of my experience on this interesting and important subject. It would appear, that much of the confusion that has existed with respect to the diagnosis of pericarditis, has arisen from not separating the consideration of that form of the disease, in which there is a copious liquid effusion, from that in which the surfaces of the pericardium are only separated by an exudation of lymph. It will be found, I think, that the general symptoms of these two varieties are often exceedingly different, and that those cases which most often prove fatal, with that assemblage of distressing symptoms noticed by all authors, more frequently belong to the first species; while the second is often, as far as external symptoms go, a nearly latent affection."

or totally absent; in which case, the practitioner must carefully employ pressure as above directed; and if, notwithstanding, no pain is felt by the patient, he must carefully turn his attention to the remaining symptoms. Should the pulse be feeble, faltering, intermittent, unequal, &c., without any apparent adequate cause, (and it is well known to practical men that such a pulse rarely if ever exists in ordinary cases without an obvious cause,) this sign,* especially if attended with the usually concomitant signs of an obstructed circulation, affords evidence of the strongest description; and the evidence is greatly augmented if there be increased dulness on percussion, indicating effusion within the pericardium.

But there may neither be pain, nor an unsteady pulse, nor its usually attendant disturbances of circulation and respiration. In this case, should the action of the heart be violent and of a bounding or jerking nature without any manifest cause,—especially organic disease of the organ; and should it be accompanied by a greater degree of fever and anxiety than can be accounted for by any other existing complaint; finally, should it be attended with certain murmurs presently to be described, the physician will seldom be wrong in diagnosticating pericarditis.† The presumption is still stronger if, when the symptoms supervene, the patient is affected with acute or subacute rheumatism,—an affection which, whether severe or mild, whether in its early or its latter stages, is, beyond comparison, the most frequent cause of pericarditis and endocarditis.

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* On it alone I saw M. Chomel found a successful diagnosis in the last stage of a typhus fever, when the symptoms were extremely complex. I have delineated the heart in fig. 61 of my Morbid Anatomy.

† The three signs above enumerated, namely, increased action of the heart, fever, and a murmur which did not previously exist, are the least number that suffice (and they are often amply sufficient) to indicate inflammation of the heart; and I shall hereafter show that we may generally decide by the nature and situation of the murmur, whether the inflammation is pericarditis, endocarditis, or both. M. Bouillaud, finding difficulty in accomplishing this (Traité, i. p. 465), adds a fourth sign as indicative of pericarditis in particular: namely, dulness on percussion. He says, "But we have seen that the embarrassments of circulation and respiration may be deficient: the question then is, to know whether, in their absence, the diagnosis of pericarditis is still possible. I answer that it is, and that dulness, with or without prominence, of the præcordial region, plus the signs furnished by auscultation, in an individual who has fever, and who previously presented no signs of organic disease of the heart, are *certain* symptoms of pericarditis" (Traité, i. p. 464). Dulness is a valuable sign, but it is often absent; yet I repeat that, in its absence, the diagnosis may still be formed by the nature and situation of the murmurs.

It was an opinion of Corvisart that the most acute cases were the most obscure, because, says he, "the attack is abrupt, the progress rapid, and the termination almost sudden." This obscurity was felt by that acute observer, because he was not acquainted with any signs of the disease on which he could depend but the feeble, unsteady pulse, the anxiety, dyspnœa, lividity and other symptoms dependent on obstruction of the circulation,—symptoms which did not always show themselves early enough to afford him data for the diagnosis before the case was hopeless. At present, however, when we are in possession of so many signs, the same obscurity does not exist. I have seldom experienced much difficulty in recognising the acute pericarditis to which Corvisart refers. The most obscure cases are those mentioned by Latham and Andral, in which a fictitious inflammation of the brain or any other organ diverts the attention from the heart, and the delirium of the patient renders it impossible to obtain information from himself. Still, when apprised that such cases exist, I should think it perhaps not impossible to provide against them. If, for instance, it were the general practice (one which I invariably pursue myself*) to place the hand on the præcordial region as well as on the pulse in every severe inflammatory or febrile affection, in the same way that we daily feel the abdomen in cases of fever, even though the patient make no complaint of it, we should seldom fail to find an inordinately increased impulse or some other anomaly in the action of the heart, which would lead us to make, by auscultation, &c., a regular and probably successful investigation for pericarditis. For there can be little doubt that the symptoms, in the cases alluded to, are in reality not absent, but merely masked by others of predominant severity.

The only remaining cause of obscurity is, inflammation of some of the thoracic viscera, particularly the pleura, the pain of which may be seated over the heart. These complications, to which Corvisart ascribed the main difficulty of detecting pericarditis, will now cause little embarrassment to those who are acquainted with auscultation.

Pleurisy may, in addition to its ordinary symptoms, be detected by dulness on percussion, beginning below and extending upwards; respiratory murmur diminishing or wholly failing in the same proportion, namely, as the fluid ascends and compresses the lung; bronchophony and bronchial respiration, the former passing into ægophony when there is a moderate quantity of fluid in the cavity; and diminution or total absence, over the dull part, of the vibratory tremour communicated to the chest by the voice. Peripneumony may, in addition to its ordinary symptoms, especially pink or rust-coloured, viscous sputa, be recognised in its first stage by crepitant râle with commencing dulness on percussion: in its second stage or hepatization, by cessation of crepitant râle and respiratory murmur; distinct dulness on percussion; bronchophony and bronchial respiration, diminished respiratory movement of the affected side, and increased vibratory tremour of the voice. Finally, bronchitis may be known by the mucous, sibilous and sonorous râles. Should none of these signs be present, the negative evidence thus obtained fixes the disease on the heart; should they be present, the diagnosis of the pericarditis must be made by a general comparison and cautious consideration of all the symptoms, especially the murmurs of the heart and the dulness on percussion ascending in the direction of the pericardium. If a doubt should remain, which it seldom will, I should recommend a treatment addressed to the heart, and at the same time suited for the pulmonary complications; for even the possibility of a mistake should not be admitted in reference to an organ, where, if the cure be not *complete*, the consequences may be irreparable.

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* I observe, that Dr. Elliotson has made an identical remark in his Lumleian Lectures—a work which I had not seen when the above was published.

The diagnosis of endocarditis, when complicating pericarditis, will be pointed out under the physical signs of the latter, and in the chapter on Endocarditis.

Signs of Amelioration. In a disease the treatment of which requires so much decision and promptitude in the practitioner as pericarditis, it is necessary for him to be thoroughly conversant with the symptoms, not only of deterioration, but also of amelioration. To these, therefore, I shall advert.

If the worst symptoms decline, namely, the feeble, fluttering, unsteady pulse and impulse of the heart, the feeling of faintness and suffocation, and the constrained position to which that feeling confines the patient, we may be tolerably sure that the fluid, on which these symptoms commonly depend, is decreasing by absorption; and the evidence is almost positive, if there be also a commensurate diminution of morbidly extensive dulness on percussion. But, notwithstanding, should pain, increased impulse, fever, anxiety, and a murmur of pericardiac attrition continue, the inflammation is in progress, is adding to the accumulation of lymph, and is possibly tending to the effusion of purulent fluid. But should the pain, instead of being fixed and pungent, become a mere diffuse uneasiness, or wholly cease; should the anxiety decrease, the murmur of attrition become inaudible, and the peculiar vehemence of the heart's action gradually degenerate into the beat of a merely accelerated circulation, the inflammation may be presumed to be on the decline; but it is not until all these symptoms have completely ceased, that it can safely be said to have terminated.

Still, lymph and adhesion of the pericardium may remain, rendering the reparation imperfect; and such we may consider to be the case if, with every advantage of perfect tranquillity and abstinence, the motions of the heart do not, in due time, completely regain their natural standard, and still more if, on very gradually returning to corporeal exercise, the patient find himself, after an adequate trial, incapable of his wonted exertions in consequence of palpitation and shortness of breath. If the case was complicated with endocarditis, and a valvular murmur remain, more or less incapability of exertion is to be expected as a necessary and permanent effect, since the patient labours under valvular disease.

Physical Signs.—Percussion. When the pericardium contains more than half a pint of fluid, the resonance of the præcordial region becomes dull over a greater extent than natural, in proportion to the quantity; and I have observed, though I know not whether others have remarked the same, that the dulness mounts higher up the sternum, in the direction of the great vessels, than when it is occasioned by mere enlargement of the heart. The impulse also, it may be added, is undulatory, and not exactly coincident with the first sound, in consequence of the heart having to displace the fluid interposed between it and the thoracic walls, before it can impinge against the latter, when the patient is in the horizontal position (Vid. Hydropericardium).

Further, the first sound and any murmurs generated in the auricular valves are more obscure than natural, in consequence of having to be transmitted through a mass of fluid and lymph: the second sound may be heard high up the vessels almost as distinctly as natural. M. Louis states that he once found a temporary effusion of fluid attended with a prominence of the cardiac region, but he is not sure that the prominence did not previously exist. I do not happen to have noticed a prominence from this temporary cause, but I think it not improbable in young subjects in whom the cartilages are flexible. Dulness on percussion is a sign of the first importance in pericarditis.

The impulse of the heart is increased, sometimes greatly:—not only heaving the thoracic walls vigorously, but being remarkable for its abrupt character: whence it often visibly shakes the whole anterior chest. Some beats are generally stronger than others, even when the action is regular. Such is the nature of the impulse so long as there is little or no serous effusion, and it is apparently attributable to an increase of irritability in the organ, resulting from inflammation. But when considerable serous effusion takes place, and by compression embarrasses the action of the heart, the impulse is feeble, faltering, irregular, and unequal. When pericarditis is attended with aortic regurgitation from endocarditis, the pulse or rather throb of the arteries, often perceptible over the whole body, is of a remarkable nature, each undulation of the blood shooting with velocity under the finger, as if through a lax or imperfectly filled tube, and constituting what is called a jerking pulse,—the pulse that we feel during reaction after uterine or other excessive hæmorrhage. Very frequently, it is accompanied with a distinct thrill. Sometimes it is stronger and more voluminous, at others, smaller and weaker; yet, in the latter case, it still retains the same jerking character.* If the injury of the aortic valves and consequent regurgitation remain uncured, the jerking pulse is permanent.

The Sounds. Two classes of murmurs, derived from two distinct sources may attend pericarditis. I shall notice them in succession.

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* When I wrote the above paragraph in the first edition of this work, I was under the impression that the jerking pulse was connected more with the pericarditis than with the regurgitation, which latter I subsequently ascertained to be its sole cause. Its connexion with this cause had not previously been noticed.

The 1st class are *direct* signs of pericarditis; for they result from attrition of the opposite surfaces of the pericardium roughened by lymph, and also, in some cases, from the roughened surfaces agitating or churning a little serum between them. The murmurs are, further, attended with a *vibratory tremour* generally perceptible to the hand. Dr. Stokes found this tremour in five cases out of six. I have noticed it several times. Dr. Watson mentions it once (Med. Gaz. April 11, 1835). These phenomena only exist, 1. at the commencement of pericarditis, before any considerable liquid effusion has taken place; 2. in cases of what has been denominated *dry* pericarditis, that is, with effusion of lymph alone; 3. in cases where the absorption of the fluid portion has at length permitted the roughened surfaces to come in contact: for it is obvious that the interposition of any *considerable* quantity of fluid, by separating the surfaces, would suspend the phenomena. In proof of this, I have notes of several cases in which, so long as a copious serous effusion was indicated by dulness on percussion and the other signs of hydropericardium, (see Hydropericardium,) the attrition murmur and tremour did not exist; but they commenced so soon as a diminution of the dulness, &c. denoted that the fluid had been sufficiently, though not wholly absorbed.

The murmur is almost always double, accompanying the two sounds of the heart, in correspondence with the movements of the organ backwards and forwards within the pericardium. I have, however, occasionally found it stronger with the first sound, and once or twice I have heard it accompany that sound exclusively. This might be anticipated, in consequence of the superior force of the systolic movement.

The murmur presents very diversified characters, which appear to me to depend on the degree of firmness and roughness of the lymph, the quantity of fluid with which it is mixed, and the greater or less violence of the heart's movements. These diversities, therefore, do not diminish the value of the sign, but they require to be severally described, lest the auscultator, from ignorance of what he has to anticipate, should wholly overlook the sign when he encounters one of its more uncommon varieties.

The murmur, then, has generally more or less of a *rough* character, sometimes like the *rasping* of wood, or the *grating* of a nutmeg; or sometimes like the rustling of silk, or even the crackling of parchment (Bouillaud). Occasionally it has a softer character, approaching to ordinary bellows-murmur. Very rarely, it resembles the creaking of a new shoe-sole. I have also heard a lower *croaking* tone, like the tearing of linen cloth; and I have likewise heard a *continuous* hollow rumble, not noticed by authors, and which I ascribe to the agitation of as large a quantity of fluid as is compatible with the production of a murmur; for in one case, in which the fluid originally caused dulness as high as the 2d rib, the rumble came on, with tremour, when the quantity of fluid became moderate; it passed into a double attrition sound, with tremour, when the fluid underwent further absorption, and both phenomena ceased when complete cessation of dulness and other signs indicated adhesion of the pericardium, which I ascertained to have taken place by post mortem examination a year and a half afterwards.*

It may strike the reader as rather incredible that so many varieties of rubbing murmur should be produced by a single affection; but his doubts will cease on finding that he may closely imitate nearly the whole, even the creaking sound, by rubbing a damp finger, with various degrees of force, and in various positions, against the back of his hand, while he listens with a stethoscope applied to the palm. From experimenting in this simple way, and from the cases which I have seen or read of, I think it probable, 1. that the rough sounds of *rasping*, *grating*, &c., are referable to firm and rugged lymph, the sound being louder in proportion as the lymph is rougher and the action of the heart stronger; 2. that the *rustling* and *crackling* sound are referable to soft and wet lymph; for I have often heard the same from friction of lymph on the pleura *immediately* after absorption of the fluid; 3. that the *softer rubbing* sound like bellows-murmur is due to soft, dryish lymph; for I have often heard the same from friction of lymph on the pleura long after the fluid had been absorbed: 4. that the *creaking* and *croaking* sounds are owing to very dry, tough lymph or granulations; for such was the state of the parts in the two cases recorded by M. Bouillaud (Traité, Pericarditis, cases 1 and 4), and, apparently, in two cases by Dr. Stokes (cases 3 and 4, Dublin Journal, vol. iv): further, the creaking sound may be imitated by rubbing together the fingers made sticky by resin, or even by dampness alone. This experiment leads me to think that deficient lubricity of the pericardium, from defective secretion in the earliest stage of inflammation, may possibly be *one* of the causes of the creaking sound, independent of lymph. This was M. Collin's explanation, and it has generally been rejected because it did not explain *all* cases; but I suspect that it will be found correct as far as it goes. 5. That the *continuous rumble* is owing, as already stated, to the churning of a little fluid.

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* Case of Robert Jones. The murmur and thrill were perceptible latest at the base of the heart, and I found that this was the only part where adhesion had not taken place. From this and other cases, I suspect that the last spot from which fluid is absorbed, is the angle between the base of the heart and the great vessels.

My main object in offering these explanations is, to render the murmurs intelligible by describing how they may be imitated. To the explanations in the abstract, whether correct or not, I attach little importance; since, provided it can be ascertained that there *is* an attrition murmur, it matters little which of the above characters it presents, and what is the particular state of parts producing it. The best proof of this is, that a murmur may pass through several or most of the above characters in the progress of a single case. The transitions may be remarkably rapid; as, for instance, when an abstraction of blood, by diminishing the force of the heart's action, and consequently the violence of the attrition, suddenly converts a loud rasping or grating sound, with distinct vibratory tremour, into a soft bellows-murmur without tremour (Stokes). Different kinds of murmur may even exist over different parts of the same heart. These transitions &c. even augment the value of attrition murmurs as signs, because they are not observed in valvular murmurs.

When the effusion of lymph is limited to a particular spot, the murmur exists at the corresponding part alone. Thus, in some cases, Dr. Stokes found the signs at the apex only; in others, merely on one side; and in one case he was able to trace the extension of the disease over the entire surface of the heart by the corresponding advances of the murmur.

The same gentleman has observed, as a corollary to the preceding paragraph, that the extent to which the sounds of attrition are propagated, is in general very limited. In by far the greater number of his cases, they were not audible beyond the actual region of the heart, and he "has often observed that, on moving the stethoscope little more than an inch from a situation where the sounds were loud, they totally ceased, although the contractions (sounds) of the heart continued distinctly audible." I suspect that this limitation of the murmurs results from nothing more than their weakness, aided, perhaps, in some cases, by their being generated on the posterior surface of the heart; for when a murmur, generated on the anterior surface, is loud, I see no reason why it should not be extensively propagated. Accordingly, in Dr. Watson's case (Med. Gaz. April 11, 1835, p. 62), the murmur, which "represented very exactly the upward and downward action of a saw on rough wood, was by far the loudest sound of the kind that he ever heard. It was distinct over the whole of the chest, both before and behind, only somewhat fainter as the distance from the heart became greater: with your ear upon either scapula, you might have supposed that you were listening to the deep buzzing vibrations of the larger string of a bass viol." This was occasioned by the pericardium being covered, except upon the posterior surface of the left ventricle, "with a thin coat of *firm*, gray lymph, quite rough with minute papillæ, projecting from almost every point of its surface, of an almost *horny consistence*, harsh and resisting to the touch, &c." Bouillaud also mentions (Case 1. of Pericarditis) a creaking sound which could be heard an inch from the præcordial region.

Adhesion of the pericardium may be inferred from three circumstances; 1st, cessation of a distinct attrition murmur; 2d, no increase of dulness on percussion, whence the cessation cannot be attributable to fluid in the pericardium; 3d, strong jogging, and sometimes double-jogging action of the heart, even though fever has subsided—a phenomena referable to the organ being bound to the spine by the adhesion (See Adhesion of the Pericardium).

Resolution may be inferred if an attrition murmur cease without leaving increased dulness on percussion, or inordinate jogging action, while all the other signs indicate resolution.

It may be inferred that neither adhesion nor resolution has taken place, if an attrition murmur continue up to the time of death.

I postpone explaining the diagnosis of murmurs of attrition from valvular murmurs, till I have noticed the latter, to which we next proceed.*

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* When the first edition of this work was published, the class of murmurs from attrition of the pericardium had not been discovered, with the exception of the "creaking of new leather," (craquement de cuir neuf,) by M. Collin, in 1824. I was criticised by Dr. Stokes in an Irish review for not even mentioning this sign. I must frankly confess that I doubted its existence; for I had searched for it in vain, and had never met with any one, either in this country or in the Parisian hospitals, who had heard it. Nor will my incredulity now appear surprising; for M. Bouillaud states in 1835 that neither he nor Andral had heard it in a pure form more than once (Traité, i. 198): Laennec, Louis, Rostan, Bertin, Latham, Elliotson, all writers on pericarditis, had not heard it; Dr. Stokes in 1834 heard it twice, and Dr. Watson in 1835 heard it twice.

The history of the discovery of the various murmurs of endo-pericarditis is as follows. After the discovery of "creaking of new leather" by Collin, in 1824, Dr. Latham, in 1826, discovered a bellows-murmur with the first sound, as a sign of *rheumatic* pericarditis. He communicated this to me in the same year; and I found, and published in the first edit., in 1831, that the murmur accompanied not only rheumatic, but any kind of pericarditis, that it sometimes attended the second as well as the first sound, that it was referable, not to the pericardium, but to co-existent endocarditis, and that it was the earliest and best sign of inflammation of the heart. Dr. Elliotson had, unknown to me, published in the previous year, that the murmur was referable to endocarditis. I can now distinctly recollect various cases in which I noticed that the murmurs were, "croaking," "anomalous," "extraordinary;" and I entertain no doubt that these were attrition murmurs: I failed to discriminate them, because, during the last ten years, not having had a fatal case of acute pericarditis, I have not had the opportunity of post-mortem verification. Had Collin given a happier name than crie de cuir neuf to attrition murmurs, I have no doubt that they would much sooner have been recognised. Though the honour of giving the first clue to this class of murmurs belongs to Collin, and though Broussais, as will presently be shown, noticed the sound like rubbing of parchment, yet the merit of satisfactorily unravelling the whole subject is, in my opinion, to be awarded to Dr. Stokes (Dubl. Jour. vol. ii. Sept. 1833). Apparently without being aware of the researches of Dr. Stokes, Dr. Watson also published, in the Med. Gaz. April 11, 1835, two cases of endopericarditis, in which he describes the *to-and-fro* sound of attrition, and perfectly distinguishes it from the co-existent valvular sound. M. Bouillaud does not appear to claim originality respecting the attrition sounds, but states that he had observed bruit de soufflet in pericarditis at a period when he was completely ignorant of the labours of Drs. Latham, Hope, and Stokes (Traité, i. 457).

The 2d class of murmurs indicating pericarditis are *indirect* signs, and afford merely *presumptive* evidence. They proceed from valvular affections occasioned by co-existent inflammation of the lining membrane of the heart. This causes the valves to become red, swollen, thickened, and sometimes studded at their free margins with granulations of lymph, denominated vegetations. These morbid conditions contract the valves, so as to generate a murmur when the blood passes through them in its natural direction: further, the contraction frequently renders them incapable of closing their respective orifices, whence a second murmur is produced by the blood regurgitating, or flowing retrograde through them. Thus the murmur may be either sin-

I shall now subjoin quotations from Collin and Broussais, to show that their observations, though so long rejected, were correct so far as they went. Collin says, "The sound analogous to the creaking of new leather has only once fallen under my observation: it was in a man who died of chronic pericarditis. The sound continued during the first six days of the disease, and disappeared so soon as the local symptoms announced a rather abundant liquid effusion into the pericardium." He then relates that M. Dervilliers, élève interne at the hospital St. Antoine, met with the sound twice. In one case, the patient left the hospital, and the result was unknown: in the other, "he made a post-mortem examination of a man who had presented the sound during the whole period of his stay in the hospital. He found a chronic pericarditis, which had occasioned the formation of thick false membranes and numerous vegetations on the pericardium and heart. Between the surface of the organ and its envelope there were only a small number of adhesions, and the cavity did not contain a drop of serum." Collin here remarks, "*Perhaps this sound will prove a constant symptom of pericarditis before the existence of effusion into the pericardium—a symptom which will be very transitory in the cases in which the disease terminates in a few days, and more prolonged when it is chronic* (p. 64)." He adds (p. 116) "I shall explain the phenomenon by the friction of the two layers of the *dried* serous membrane. This kind of *dryness* seems to be the first effect of inflammation on the membranous tissues." (On the various modes of exploration of the chest. By V. Collin. Paris, 1824.)

Dr. Stokes says that Broussais is the only author that he can find, subsequent to Collin, who brings his own experience to bear on the subject. Speaking of the symptoms of inflammation of the heart, he says, "There is a phenomenon worthy of attention, to which enough has not perhaps been given: it is the sound of parchment, which is very perceptible by means of the stethoscope. On exploring with this instrument in commencing pericarditis, the sensation is experienced which would be given by two dry bodies, as parchment, rubbing against each other; and this sign, when conjoined with pain and distress (angoisse) can leave no doubt as to the existence of the inflammation" (Commentary on the Propositions of Pathology, 1829, vol. i. p. 398).

The number of individuals who, though unacquainted with each other's labours, have contributed to the discovery of the murmurs of endopericarditis, must afford convincing evidence, to the incredulous, of the reality of these murmurs.

gle or double, that is, may accompany either one or both sounds. The murmur with the first sound may proceed either from contraction of the aortic valves, or regurgitation through the mitral; or from the corresponding affections on the right side of the heart—which, however, I have found very rare. The murmur with the second sound I have almost always found to proceed from aortic regurgitation. For the mode of ascertaining which is the particular valve affected, the reader is referred to *Disease of the Valves, Physical Signs:* also, to p. 90.

I think that these valvular murmurs from endocarditis are entitled to the rank of presumptive signs of pericarditis, because I have found them to exist in the immense majority of cases of the latter affection. In the first edition of this work, I went so far as to say that "I had never found them absent when the heart presented an increased, jerking impulse" (p. 110); but I now think that I sometimes inadvertently included amongst them attrition murmurs, with which I was not then acquainted. Subsequent writers, however, have confirmed my observation above quoted almost in its full extent. Thus Dr. Watson says, "If I cannot affirm that the internal membrane is *always* affected, I believe that it very seldom (perhaps never) escapes. My reasons for thinking so, are, 1. the deep blowing sound (which, *in these cases*, I hold to denote an affection of the inner membrane) is rarely (perhaps never) absent" (Med. Gaz. April 11, 1835, p. 64). M. Bouillaud speaks almost as strongly. Now if endocarditis so frequently attends pericarditis, the valvular murmurs, which are *direct* signs of the former, must be valuable *indirect* or presumptive signs of the latter: and I am desirous of strongly drawing the reader's attention to their value, because they have, of late years, been too much depreciated in consequence of the absorbing attention which has been paid to attrition murmurs; whereas I feel as confident now as I expressed myself in the first edition of this work, that the valvular murmur is the physical sign which most frequently yields the first intimation of inflammation of the heart. Dr. Watson more recently expresses a similar opinion: "This *to-and-fro* (attrition) sound," says he, "is not the sound which is most commonly heard in the outset of these cases of rheumatic carditis: it is the deeper blowing sound or whiz which we hear, and which excites all our anxiety to save the patient from that mischief, the commencement of which it indicates; namely, alteration of the valvular parts of the heart" (Ibid. p. 63). The reason why the valvular murmur gives early intimation of pericarditis more frequently than the attrition murmur, I believe to be this:—pericarditis, in the majority of cases, is attended with copious serous effusion almost from the first, which prevents the developement of the attrition murmur; whereas the same pericarditis is, in the immense majority of cases, attended with endocarditis, which almost necessarily produces a valvular murmur: hence the latter murmur occurs in the majority, and the attrition murmur in the minority of cases.*

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* M. Bouillaud has not done justice to my account of the valvular murmurs occurring in pericarditis. He says that I had only a "*glimpse*" (entrevue) of the influence exercised by endocarditis in producing the bellows-murmur," and he represents me to have stated "that the bellows-murmur which takes place during *the diastole*, is the only one which should cause us to suspect this complication, so common in pericarditis." How totally he is mistaken will appear from the following passages in my first edit. p. 110. "This sign (the bellows-murmur with the first sound) was first noticed by Dr. Latham, who pointed it out to me at St. Bartholomew's Hospital in 1826. Since that time I have never found it absent when the heart presented the increased, jerking impulse. Dr. Latham restricts his observation to rheumatic pericarditis: to myself the phenomenon has appeared to exist equally in every form of the disease. Not the ventricular systole only, but occasionally, though by no means always, its diastole likewise, is attended with the bellows-murmur: and I have found this supersede, and, as it were, annihilate the natural second sound more completely in pericarditis, than, I think, in any other affection of the heart. Sometimes, in short, it is a pure whizzing equally prolonged as, and almost continued into, the first sound."

I offered two explanations of the murmur with the ventricular systole. I thought it "*probable*" (a stronger term is not used) that it was mainly referable to the morbidly abrupt contractions of the heart, &c., as occurred in dogs repeatedly bled. This explanation proved to be incorrect. The second explanation I did not offer as probable, but as certain:—"I believe that it (the murmur with the ventricular *systole*) may, in some instances, originate partly in another cause: namely, *constriction of the arterial orifices consequent on inflammation of the lining membrane.* For, as this membrane is more liable to inflammation where it constitutes the valves, than elsewhere, it is consistent with analogy to suppose that, by its intumescence and loss of elasticity, the orifices will undergo the constriction alluded to." Surely I had here more than a "*glimpse*" of endocarditis, and it was the murmur with the systole, and not with the diastole alone, as M. Bouillaud states, that indicated it. Again, "The murmur accompanying the *second* sound, I am inclined to attribute perhaps *entirely to the same constriction.* This I infer, because I have not found it produced, in any appreciable degree, by abrupt jerking action of the heart in reaction from loss of blood, and in nervous palpitation: and because, *when I have noticed it in pericarditis, I have invariably found it connected with a more or less thickened state of the*

Diagnosis of Valvular from Attrition Murmurs. Some writers, especially M. Bouillaud (tom. ii. p. 211), have experienced great difficulty in discriminating these two classes of sounds. I cannot say that, since I became acquainted with attrition murmurs, I have participated in this difficulty—even when the two classes of sounds existed simultaneously, and each was double. This is mainly from attending to the rules which I have so often inculcated; namely, of listening to murmurs of the sigmoid valves two inches or more up the aorta or pulmonary artery, where attrition murmurs are mostly inaudible; and of listening to murmurs of the auricular valves a little above the apex of the heart, where they are sure to be the loudest, whereas attrition murmurs may be louder at other parts of the heart where they happen to be generated. Further, attrition murmurs present the following distinctive peculiarities—

1. They are usually of a much rougher quality of sound than the valvular, so that, when the two co-exist, the one may be heard *through* the other.

2. When a murmur with the second sound is rough, as rasping, creaking, croaking, &c., it is certainly from attrition; as I have never known a valvular murmur with the second sound to be rough, the diastolic currents being too feeble to produce roughness (See p. 83).

3. Attrition murmurs are almost always attended with vibratory tremour; whereas valvular murmurs rarely present this phenomenon, and generally in a slighter degree.

4. Attrition murmurs are apt to undergo frequent and sudden changes of character and of situation (Stokes), which are very pathognomonic, because valvular murmurs change little in character, and not at all in situation.

Signs and Diagnosis of Chronic Pericarditis.

General Signs.—When acute pericarditis runs on unsubdued beyond ten days or a fortnight, the full limits of its ordinary

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valves. Should this be found true, the bellows-murmur of the second sound renders the prognosis more gloomy; as it bespeaks a more extensive inflammation, and the probability of subsequent valvular disease." Again, I said at p. 115, "When there is inflammatory constriction of the orifices, a murmur will attend *both* sounds."

duration, it becomes what is called *chronic*. The same name is given to the disease when, from the first, it runs a slow, insidious course, without marked or violent symptoms.

The general signs of chronic pericarditis are, in their nature, much the same as those of acute, but they are less in degree. Thus, the fever, instead of being of the smart, inflammatory kind, is more that of hectic or marcor, because there is usually suppuration or irritative emaciation; but there are occasionally active exacerbations, when, perhaps, the inflammation becomes sub-acute. The anxiety and restlessness, though sometimes great, are comparatively supportable. The position is less constrained, and I have observed that, when there is much fluid in the pericardium, the patient often prefers the sitting posture with the body inclined forwards. The circulation is less embarrassed, and the action of the heart, in the absence of adhesion of the pericardium and hypertrophy, is usually somewhat feeble, except during any temporary exacerbation of inflammatory action. It is sometimes not very irregular, intermittent, and unequal, though the pericardium be full of fluid; which I attribute to the elasticity of the membrane not being so far destroyed by the inflammation as to prevent it from gradually undergoing extension, and accommodating itself to its contents; whence compression of the heart by the fluid is in some degree obviated. The patient, I have thought, more frequently complains of a load and fulness, "something which he cannot get down," in the scrobiculus cordis, in chronic, than in acute pericarditis. In a considerable number of chronic cases, I have found œdema of the legs to occur: once within a month, in others, later. Louis saw it in two cases of less than six weeks duration. This is an important general sign, as indicating an obstacle to the general circulation.

This inferior degree of violence in the symptoms renders chronic pericarditis, especially if such from its commencement, more obscure than acute. I have, in former years, when auscultation was little known, seen it overlooked more than once. But these cases, when I now revert to them, appear to me to have presented sufficiently characteristic symptoms. The history affords great light. If the patient, previously exempt from disease of the heart, has become affected with its symptoms, attended by marcor and some degree of fever, within a period seldom extending beyond a few months, and which he often dates from a blow or fall on the breast, a rheumatic fever, or an inflammation with pain in the præcordial region, chronic pericarditis may be strongly presumed; and if these symptoms coincide with the physical signs of fluid in the pericardium, or with attrition murmurs indicating lymph, the existence of the malady may be regarded as certain.

Physical Signs.—The impulse is, cæteris paribus, weaker than in acute pericarditis without effusion, because, the inflammatory irritation being less active, the movements of the organ are less violent. If there be hypertrophy, which is apt to supervene after the lapse of two or three months, the impulse will sustain a corresponding augmentation of force; and if there be adhesion of the pericardium over any considerable extent, it will be more or less jogging, as well as strong.

The sounds will vary according to circumstances. They may be natural, provided there be neither dilatation, which augments them, and shortens the first; nor attrition of lymph within the pericardium, which creates an attrition murmur with one or both sounds, and a vibratory tremour; nor inflammatory constriction of the orifices, which may produce a bellows-murmur with one or both sounds (see p. 171), and a jerking pulse if there be aortic regurgitation (p. 165).

The signs of fluid in the pericardium are the same as in acute pericarditis (p. 164); namely, the extensive dulness on percussion, and the undulatory impulse.

SECTION III.

CAUSES OF PERICARDITIS.

THE most frequent causes are, blows, wounds, punctures,* or excessive pressure on the præcordial region, inflammation propagated from the lungs or pleura, and, *far above all, acute rheumatism.* From this cause, children and young persons, that is, those between the ages of eight and thirty-five, suffer much oftener than others—a fact which I have ascertained almost numerically on a vast number of cases. The remaining causes are, those of inflammation in general; viz. cold, febrile excitement, &c. M. Bouillaud assigns the same causes; for he says, "Of the exciting causes, the most powerful, frequent, and consequently that which it is the most important thoroughly to understand, is, incontestably, a quick and sudden chill, following a great heat, with copious perspiration, of the body, and more or less violent and fatiguing exercises" (Traité, i. 169 and 171). In the next sentence he resolves this cause mainly into rheumatism: for he says, "Hence, we ought not to be surprised if pericarditis is so commonly the companion, I do not say of pleurisy and peripneumony only, but also of hyper-acute rheumatism of the joints. *Who knows not, in fact, that this latter inflammatory fluxion has precisely for its principal cause, the vicissitudes which I have just specified?*" He then intimates that Corvisart had some idea of the connexion between rheumatism and pericarditis; since that illustrious author says, "I am disposed to regard as a frequent cause, amongst others, of this adhesion (of the pericardium), rheumatic and gouty affections." He employs the remainder of the chapter in attempting to show that the connexion in question "had been almost entirely overlooked in the great majority of cases up to the present time." He consequently inculcates it as a novel doctrine, and, to corroborate his opinion, does me the honour of a quotation to show that I was fully acquainted with it. I have not, however, the slightest pretension to originality in this idea; since, at the time when I wrote, there was not a better established doctrine in the London schools. The history of the discovery was as follows. It appears to have been first made by Dr. Pitcairn in 1788. This physician being too modest to publish, Dr. Baillie did it for him in the second edition of his Morbid Anatomy, in 1794. The connexion in question was noticed by Sir David Dundas in 1808; also by Dr. Wells, and by Dr. Odier of Geneva. Since then, it has been noticed by every modern writer on pericarditis in this country; for instance, Drs. Latham, Abercrombie, Elliotson, Davis of

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* M. Desclaux has produced pericarditis and endocarditis by piercing the pericardium and heart with needles. M. Renauldin relates a case of the same in the human subject—an individual who had made several attempts at suicide (Bouillaud, Traité, i. p. 649, note).

Bath, and myself. I deem it unnecessary to offer evidence in substantiation of a doctrine which I consider to be established beyond the possibility of contradiction; but I may state as a striking fact, because derived from a great number of observations, that acute rheumatism had preceded, in about three fourths of the worst cases of valvular disease and adhesion of the pericardium, which have occurred amongst upwards of 10,000 hospital patients, whom I have treated during the last four and a half years. The following statement of M. Bouillaud is also striking and important: "Such, according to my experience, is the frequency of pericarditis in rheumatic individuals, that one might affirm, *a priori*, that, out of twenty patients affected with universal acute rheumatism of the joints, accompanied with smart fever, *the half, at least*, would present symptoms of pericarditis or of endocarditis, and often, of these two inflammations united" (Traité, i. 472). This statement would appear incredible to non-auscultators, because they must almost necessarily overlook those cases —especially of endocarditis, which are scarcely revealed except by physical signs; yet, according to my own observation, the statement is not wide of the truth when rheumatic affections are neglected or inefficiently treated.

The extension of rheumatic inflammation to the heart or dura mater, (of which latter, however, I have never seen a distinct instance,) was formerly regarded as a *metastasis*, that is, a change of seat—a total desertion of the external parts and a concentration of the disease on the internal organ. But I have no hesitation in expressing my unqualified conviction that this idea is erroneous—an obsolete relict of antiquated ignorance; for I have not only, in cases innumerable, seen the heart attacked while the rheumatism existed in full intensity in the joints, but I have seen the heart attacked before the joints, and I know that other practitioners have seen the same. Dr. Charles of Putney lately (January 1828) favoured me with one instance. Hence, I believe that what has been called *metastasis*, is nothing more than an extension of the inflammation to the *internal* fibrous tissues, namely, of the peri- and endo-cardium, of the dura mater, sclerotica, &c., precisely as it extends or migrates from the fibrous tissue of one joint to that of another, by what Bichat has happily denominated the "affinity of tissue."*

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* Reflecting on what has now been said above, relative to the frequency of peri- and endo-carditis in acute rheumatism, and to the frequency of incurable valvular and pericardiac disease as sequels of the peri- and endo-carditis, are we not driven to exclaim, how important, how fearful a disease is acute rheumatism!—an affection till lately,—in short, till auscultation threw light upon it, considered painful and troublesome, indeed, but harmless to life! I have made brief notes of between two and three hundred cases, and observed many more, with the view of ascertaining *by comparison* which was the most successful mode of treatment. The results are published, from a lecture by the writer, in the Med. Gaz. February 25th, 1837; but, considering the magnitude of the subject, it may not be irrelevant here to glance at them.

The most successful treatment beyond comparison, was a modification of that introduced fifty years ago by Dr. Hamilton of Lynn Regis, who, in addition to bleeding and purgatives, excited salivation by calomel and opium. The modification to which I allude, and which merely consists in avoiding salivation, I first saw employed by my colleague Dr. Chambers, in St. George's Hospital. Leaving the merit of the plan to him, I shall offer my own experience of it in about 200 cases of acute, and active chronic rheumatism.

1. *In acute rheumatism.* After one full bleeding, or even two in robust subjects, but without any bleeding in the feeble and delicate, I give, every night, gr. vii of calomel with 1¼ of opium, or gr. x of calomel with gr. ij of opium, according to the age and the severity of the symptoms. This is followed every morning by inf. sennæ c. ℥iss, magnesiæ sulph. ʒij, and mannæ ℥j, which should act at least four or five times. In addition, (though this is not a part of Dr. Chambers' plan,) I generally give the following draught thrice a day, as it has appeared to me to expedite the cure—partly, perhaps, by the additional opiate, and partly by the sedative effect of the colchicum. ℞ vini colchici, m xv ad xx; pulv. ipecac. comp. gr. v; mist. salin. ʒx; syrupi, ℥j ♏ft. haustus.

When the pain and swelling are greatly abated, if not almost gone, (which often happens within two days, and almost always within four,) I omit the calomel, or, if the gums become in the slightest degree tender, I omit it even earlier. The opium, I continue to the extent of gr j or iss at bed-time, and in severe cases I add a grain at noon,—for, without an anodyne, the pains are apt to recur. I also continue the colchicum draughts and the senna draught.

No local treatment is necessary beyond warm or cold applications, according as the patient finds them agreeable.

If the patient is not well in a week, I consider it a case of exception; and the exceptions are generally in those who are subject to rheumatism, and who, therefore, usually have it in a more obstinate, chronic form. The advantages of this plan are, 1. The patient is generally well, sound, and fit for work in a week or ten days after the pains have ceased. 2. The gums are rarely affected—especially if it be previously ascertained that the patient has not a peculiar susceptibility of the action of mercury. 3. *It is rare to see inflammation of the heart supervene,* if the treatment is early commenced: I think that about one case in twelve would be the maximum in my practice. 4. If the slightest symptoms of pericarditis or endocarditis do supervene, a few additional doses of calomel and opium, (as gr. v of calomel with gr. j of opium every four or six hours,) will generally affect the constitution in 20 or 30 hours,

SECTION IV.

PROGRESS AND DURATION, TERMINATIONS AND PROGNOSIS OF PERICARDITIS.

Progress of Pericarditis. The progress and duration of this, as of most other inflammations, varies according to circumstances. If intense and extensive, and especially if complicated with severe endocarditis or pleuritis, it may be fatal within 30 or 40 hours. Andral relates a case of rheumatic pericarditis, without other complication, which terminated in 27 hours (Clin. Med. iii. p. 416. 1826).

I have not treated a case during the last ten years, on the plan which, with two or three cuppings or leechings on the præcordial region, almost always places the patient in a state of safety. I have never lost a patient by rheumatic inflammation of the heart since I have employed this plan, and I have been told by other hospital physicians that they have been scarcely less successful.

2. *Active chronic rheumatism.* Here calomel and opium may be given in smaller doses, as gr. v of calomel and gr. j of opium, every night; but they require to be continued for a longer time, as five or six nights. Care should, however, be taken to stop short of ptyalism, especially in the scrofulous. The other particulars of the treatment are the same as in the acute form. Local treatment, however, is more beneficial than in the latter: namely, the bleedings, if necessary, may be local instead of general, and blisters, liniments, plasters, &c. may ultimately be employed if a joint continues obstinately affected.

I cannot doubt that the opium contributes importantly to the cure—perhaps by allaying pain, and thus diminishing the irritative fever dependent on it: or, possibly, by modifying in some unknown way the vital constitution of the blood. However this be, I have assured myself of the fact that opiates and purging alone, will cure many cases of acute rheumatism remarkably well. Others have used different narcotics with similar success. My friend, Dr. Lombard of Geneva, states that he has had remarkable success with the spirituous extract of aconite, in doses of gr. half, gradually increased to gr. ij or even iij, every three hours. I have also heard that ℈j of conium daily, in divided doses, has produced good results.

M. Bouillaud has lately extolled, and introduced to his countrymen, apparently as a novelty, the plan of copious and frequent bleeding at short intervals for acute rheumatism. This plan, which is as old as Sydenham, and which I saw carried to its very utmost limits, in Scotland, nearly 20 years ago, is not to be compared in efficacy with the plan above described, either as a prompt means of curing rheumatism, or an effectual mode of preventing inflammation of the heart; while it has the disadvantage of exceedingly reducing the strength and rendering convalescence very protracted. I readily admit, however, that I have seen many cases promptly and effectually cured by this plan.

alluded to in the last note and presently to be fully described, which did not terminate favourably in a week or ten days, and often much less. There may, however, be exceptions, and I ascribe it to good fortune that they have not yet occurred to myself.

Chronic pericarditis, once established, may run on several weeks, or, if neglected, several months. I have notes of three or four such cases.

Terminations of Pericarditis. Resolution is the most common. The effused fluid and lymph are absorbed, but a little lymph frequently remains, eventually constituting white spots of cellular tissue, and sometimes forming the groundwork of cartilaginous and osseous transformations. Authors also mention granulations and vegetations, but I do not happen to have met with them. All these depositions are sometimes rough, and they may then create an attrition-sound: but authors seem to be agreed that, with the exception of extensive osseous or cartilaginous depositions, they are not incompatible with perfect health. I have not had opportunities of forming a confident opinion.

Adhesion of the pericardium is a less favourable termination. The adhesions are sometimes partial, and, if not pervading a considerable surface, they may become long and loose by extension, —in which case they offer little or no impediment to the action of the heart, and are consistent with the enjoyment of perfect health. In other cases, they are universal, and they then, according to my experience, offer an important, and in most instances an ultimately fatal impediment to the action of the heart. This subject will be fully noticed in the section on Adhesion of the Pericardium.

Chronic pericarditis is an occasional termination of acute, if the latter be neglected or inefficiently treated, and especially if this occur in a scrofulous or otherwise unhealthy constitution.

Valvular disease is a frequent termination of the endocarditis accompanying pericarditis. It may be ascertained to exist by one of the valvular murmurs described at p. 171, and by palpitation on exertion, continuing after the fever and other inflammatory symptoms have subsided. If there happen to be aortic regurgitation, the pulse will be eminently *jerking*, and this must not be confounded with the *hard* or *sharp* pulse, and lead the practitioner into the gross error of supposing that it results from the persistence of fever or inflammation.

Prognosis of Pericarditis. Before the diagnosis of pericarditis was redeemed from deep obscurity by the light of auscultation; and, it may safely be added, before the use of mercury was understood in the treatment of acute inflammations in general, pericarditis was one of the most dangerous and destructive diseases in the nosology. The acute and chronic forms were supposed by Corvisart to be necessarily fatal, what he denominates the sub-acute form alone affording a hope of recovery. This statement, however, must now be admitted with limitation, since it is certain that Corvisart and his cotemporaries must necessarily have overlooked many of the slighter cases, which underwent resolution.

At the present period, I should venture to say that, when the treatment has not commenced at too late a period, the prognosis of acute pericarditis with respect to life, is decidedly favourable. Even M. Bouillaud, who does not employ mercury, and who, as already stated, has introduced to his countrymen the system of free bleeding as a supposed novelty, says, "What I can affirm for my own part, is, that I have cured *the greater number* of cases of pericarditis that I have met with for some years; and the truly extraordinary success which I obtain every day from blood-letting carried to a greater extent than is commonly done, in acute inflammations in general, affords me a well-founded hope that the majority of cases of pericarditis to which this method is well applied, will not resist it. The most rebellious,—the most destructive, will be those complicated with intense endocarditis or very violent pleurisy or pleuro-peripneumony" (Traité, i. p. 476).

The plan of M. Bouillaud, in all its details, has been universally in vogue in this country, (where dread of the lancet has seldom been a national sin—though I would not say so much for the converse proposition,) during a period extending far beyond my recollection; nor has its success, according to my observation, been less than he represents—namely, a restoration of "*the* *greater number.*"* But this amount of success I consider to be *very unsatisfactory*, and I wish to express myself strongly on this point, in order to draw attention to the use of mercury as an adjunct to the antiphlogistic treatment. It has already been stated that I have not lost a case of acute pericarditis during the last ten years, though my opportunities as physician, successively, to two of the largest hospitals in London, have not been limited. I have understood that Dr. Latham, physician to St. Bartholomew's, did not lose a case for a nearly similar period, till last year, when he lost two; and Dr. Watson, physician to the Middlesex Hospital, informed me that he also had lost only two or three in the same time. Hence it appears that the mortality ought to be far less than that stated by M. Bouillaud. I cannot numerically estimate the exact proportion, but I should think that one fatal case in a dozen would be the outside. I do not mean, however, that adhesion of the pericardium, or ultimate valvular disease from concomitant endocarditis, would be prevented in the whole number of cases specified. In fact, the valvular affection is exceedingly difficult to obviate: some say that they seldom succeed: I have certainly been more fortunate, and this I ascribe to my practice of pursuing an equally vigorous treatment for it, as for the worst cases of pericarditis, and continuing the mercurial and counter-irritant part of the plan for a much longer period, as will hereafter be explained.

With respect to the prognosis when valvular disease is established, it is more or less unfavourable according to the situation and extent of the affection; but as the particular consideration of this is long, the reader is referred for it to the chapter on valvular disease.

The ultimate prognosis is unfavourable, as already intimated, when adhesion of the pericardium has taken place; and still more so when, from bad diagnosis or inefficient treatment, pericarditis with much effusion has become chronic.

In reference, therefore, to these three last terminations, and to the fact that their prevention, or, in other words, the possibility of effecting a *complete* cure, is limited to a very brief period and supposes a high degree of diagnostic and practical skill in the practitioner, endopericarditis must still be regarded as one of the most formidable diseases incident to the human race and worthy of the deepest study of the physician.

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* In another part (tom. i. p. 480) he says, "Almost all the cases of pericarditis with which I have met." I am at a loss what to understand from these discrepant statements.

SECTION V.

TREATMENT OF PERICARDITIS.

*Treatment of Acute Pericarditis.**—The antiphlogistic treatment, in as energetic a form as circumstances will allow, should be employed with the utmost promptitude. The loss of a few hours at first, may be irretrievable, and hence hesitation and indecision may seal the fate of the patient. If the attack is recent and the patient's strength will admit, blood should, in the first place, be drawn freely and by a large incision, from the arm of the patient in the erect position, so as to bring him to the verge of syncope. From five and twenty to forty leeches, according to the strength, should then be applied to the præcordial region so soon as the faintness from the venesection disappears and reaction commences,—which generally happens in the course of from ten minutes to an hour or two. Unless the pain be completely subdued by these measures, the leeching, and in some cases the general bleeding also, may be repeated two, three or more times, according to the strength, at intervals of from eight to twelve hours; or, what is a better rule, so soon as the pulse and action of the heart denote a recommencement of reaction.

It is not, however, in every case, that so active a treatment is required. I have seen a single prompt and abundant application of leeches or a cupping at once subdue every formidable symptom. When the patient, either from age, a feeble constitution, or the advanced state of the malady, cannot bear extensive depletion, local bleeding is, according to my observation, decidedly preferable to general: but it should be practised effectually,

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* Laennec did not even mention the treatment. MM. Bertin and Bouillaud, in 1824, recommend, in general terms, general and local bleeding, rigorous abstinence and complete repose. Also counter-irritants for the chronic form.

—by cupping to twenty ounces or more, or by the application of from twenty-five to thirty or forty leeches. When, from depletion having already been carried to a great extent, or from the advanced stage of the disease, it is not safe to draw much more blood, yet it appears expedient, from the persistence of pain, &c. to draw some, I have generally found that a smaller quantity drawn by cupping produced more effect than a larger by leeching. The cause of this probably is, that, by cupping, it is drawn more expeditiously.

I may finally remark that, though blood ought to be drawn with all the vigour that I have described when the usual indications for its emission exist; yet, in cases where mercury is employed, as presently to be described, those indications so soon cease, from the controlling power of this remedy, *that the total quantity of blood lost will rarely be considerable.*

While the bleeding is in progress other means should not be neglected. The intestinal canal, if at all confined, should immediately be evacuated by a purgative enema. Three drachms of senna leaves and an ounce of sulphate of soda infused in a pint of boiling water, and strained, answers the purpose. If infusion of senna is not at hand, a scruple of comp. extr. of colocynth may be substituted. At the same time, five grains of calomel with five or ten of comp. extr. of colocynth, and two or three of extr. of hyoscyamus, should be given, and, in two hours, be followed by a senna draught.

The strength of the remedies employed must in each case be apportioned to the vigour of the patient's constitution, but the object is the same in all—expeditiously to prostrate the action of the heart, and for a time to keep it prostrate by preventing the re-establishment of reaction. If this object can be accomplished for the first twenty, thirty, or forty hours, the disease frequently does not rally, but remains perfectly under the control of remedies. I feel satisfied that *a degree of activity in the first instance, which to some may appear excessive, is an ultimate source of economy to the strength of the patient; for the disease is subdued at once, and the protracted continuance of depletory measures, the most exhausting to the constitution, is rendered unnecessary.*

In addition to the above measures, diluent, cooling drinks, as four scruples of supertartrate, or two of nitrate of potass in a quart of water and flavoured at pleasure, should be allowed in unlimited quantity, in order by diluting the blood to render it less stimulant to the heart. Nauseating doses of tartrate of antimony, as one-sixth to one-eighth of a grain, every two hours, may be employed with advantage. The diet should consist wholly of the weakest slops, as barley water, gruel, weak tea, arrowroot. &c.

But the antiphlogistic treatment alone is not to be relied upon: rarely does it, in a severe case, effect a complete cure. The practitioner sees all his resources gradually exhausted, while the disease proceeds with an even, uncontrolled tenor, to its fatal termination. Sometimes, indeed, all the other symptoms disappear, but the action of the heart remains stronger than natural: at other times the heart even regains its healthy action and the cure appears complete; yet, in both these cases, the palpitation, accompanied with symptoms of organic disease of the heart, recurs when the patient resumes his accustomed occupations. The reason of this is very intelligible. Unless the effused lymph, as well as the serum, be absorbed, it causes an adhesion of the pericardium, and thus constitutes a destructive disease; or, if the pericarditis was complicated with endocarditis, an irreparable valvular lesion is its sequel. Now antiphlogistic measures can neither prevent the effusion of lymph, nor *with any degree of certainty* cause its absorption. Mercury *can* do this,—as is visibly displayed in iritis, and as has been proved in this country by an overwhelming amount of irrefragable evidence, ever since the mineral was introduced as a remedy for acute inflammations by Dr. Hamilton of Lynn Regis, in 1783. Mercury, therefore, is the sheet anchor of the practitioner. Dr. Latham is of opinion that its success is restricted to the condition of its producing salivation, and producing it rapidly. Though, from many observations, I am satisfied that there is an advantage in promptly producing a decided effect; and though, therefore, I always aim at this by administering full and frequent doses at first; yet I do not think that success is *restricted* to the condition of salivation, or even of a sensible effect on the gums being produced; for I have frequently seen cases in which cures, not falsified after many months, were effected, though salivation was not produced. The mineral, however, was freely administered, and probably produced its specific effect though not in an apparent manner. If, therefore, there be a distinct and decided suspension of the symptoms before the gums are touched, I do not hesitate to diminish or even omit the mercury, as I am never willing to push the remedy beyond what is barely sufficient to subdue the disease. From five to eight grains of calomel, or, as less irritating for delicate bowels, from ten to fifteen of blue pill, prevented from purging by a grain or a grain and a half of opium, three times a day, commencing after the first bleeding and a purgative, generally produce the effect with sufficient expedition. When greater promptitude is required, ten grains of calomel with two of opium may be given at the first dose, and three grains with half a grain of opium, every three hours afterwards. Inunction may be superadded, or, if even the milder preparations, (pil. hydrarg. or hydrarg. cum cretâ,) taken internally, irritate or purge, it may be partially or wholly substituted. Any quantity between ʒij and ℥j of the ung. hydrarg. fort. may be rubbed into, or smeared on the arm-pits and groins, night and morning; but, to avoid unnecessary salivation, the latter quantity should not be used more than three or four times, unless it be wholly substituted for the internal preparations. A manifest abatement of the symptoms generally takes place immediately on the effect of the remedy becoming apparent in the mouth—especially if a decided soreness is established within the first thirty or forty hours. A tender state of the gums (for more is scarcely necessary) should be maintained for a week or ten days or even longer, unless the symptoms completely yield before the expiration of this period.

Should pain continue in the advanced stages of the malady, and after the period for applying leeches has passed, blisters may be resorted to, and repeated in quick succession, with great advantage. I have occasionally found a third or a fourth necessary before the pain has been completely removed. They are equally useful in cases of considerable effusion, the absorption of which they greatly promote.

In the repetition of blisters, as well as of leeches, cupping, and venesection, and in the selection of one of these remedies in preference to another, much must necessarily be left to the judgment of the practitioner. It is only experience which can teach the exact adaptation of remedies to the circumstances. It must also be left to his discretion whether to give sedatives or not. When the restlessness and nervous irritability were great, I have seen much benefit derived from tinct. hyoscyami m xv ad xx with the same quantity of tinct. digitalis, in a draught three or four times a day. Sedative remedies, however, should not be given until the first severity of the inflammation has subsided; nor should they ever be allowed, by producing their poisonous effects, to confuse the symptoms, already sufficiently complex, in the latter stages.

During convalescence, it is sufficient to say that a spare, unstimulating diet and extreme tranquillity must be imperatively enjoined until the action and sounds of the heart have become perfectly and *permanently* natural.

An individual who has recently been affected with pericarditis is very liable to a recurrence of it: especially if it has resulted from acute rheumatism and the reparation has been incomplete. In this case, should the rheumatism return, it rarely fails to be accompanied with a renovation of the pericarditic symptoms. This cannot be a subject of surprise; for it is consistent with general analogy that a part recently injured by inflammation, is more susceptible than a healthy tissue of inflammatory action—the reason of which probably is, that the vessels of newly organised adventitious structures are more tender and irritable than others. Secondary inflammation, however, has not the same energy and intensity as that of a healthy structure, it yields more promptly to curative measures, and is more completely within the powers of medicine. Hence a first attack of pericarditis is more dangerous than any subsequent one. It is comparatively rare for a patient to die from the direct effect of a recurrent attack; and, what is still more remarkable, he may sustain several without being left in a materially worse condition than after the first.

Much discretion, however, is requisite on the part of the practitioner to bring such recurrent attacks to a favourable termination, and the danger of doing too much, is perhaps greater than that of doing too little. He must, in particular, be cautious of bleeding too extensively with the object of reducing the excessive energy of the heart's action; for this energy, he must recollect, is a consequence, not of the inflammation only, but partly also of an organic affection of the organ, (viz. hypertrophy or valvular disease,) left by the primary attack. Nor is there the same motive for a vigorous employment of mercury; for, the heart being already irreparably disorganised, it would be chimerical to entertain the expectation of effecting a *perfect* cure. The object, therefore, should be, simply, to prevent deterioration by combating the inflammation as it presents itself.

For the accomplishment of this object, a moderate use of bloodletting and mercury suffices; and leeching or cupping on the præcordial region is more efficacious and less exhausting than venesection. Blisters are, in these cases, peculiarly beneficial, and they may be repeated in quick succession, on different parts of the præcordial region, as often as they are required and can be borne. When there still remains a little lingering pain, which scarcely authorises vigorous measures, but cannot prudently be left, the most valuable and convenient remedy has appeared to me to be, a plaster composed of a scruple of potassio-tartrate of antimony, four scruples of the emplast. picis comp. and two scruples of wax to diminish the tenacity of the adhesion.

In these cases, also, where the sufferings of the patient, though perhaps not severe, are very protracted, and accompanied with much loss of rest, great advantage is derived from a pill of from three to six grains of extr. of hyoscyamus at bed-time, and moderate doses of tincture of digitalis during the day, the specific poisonous effect of the latter remedy being obviated by omitting it for a couple of days after every three or four. General dropsy may occur if there be much impediment to the circulation, and it must be combated with the usual diuretics.

Treatment of Chronic Pericarditis.—When pericarditis is essentially chronic, and the cavity appears to contain fluid, counter-irritant remedies are the most suitable. After what has already been said, it will be sufficient merely to mention blisters, either in succession or kept open with savine cerate, the tartrate of antimony and pitch plaster, and likewise issues and setons. The last remedy, however, generally creates so much irritation as to do more injury by deteriorating the general health, than good, by its local effect. Mercury to a moderate extent, may, if discreetly employed, be advantageous by promoting absorption; but, in general, the patient is too much reduced by constitutional irritation, to admit of more than the mildest action of this remedy. If general dropsy supervene, as I have seen happen in several cases, diuretics must be used in the usual manner (See Diseases of the Valves, Treatment).

The diet may, in chronic cases, be more nutritious, comprising light animal food and broths.*

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* The reader will be desirous of knowing the treatment, which M. Bouillaud has proposed as new and pre-eminently efficacious. The following is his own account.

"Like all the other inflammations in general, acute pericarditis requires the use of bleeding, repose, low diet, demulcent and refreshing drinks, and of emollient applications. Hitherto, this mode of treatment has very rarely been completely followed out, principally because pericarditis has often been overlooked, or recognised too late. Now that the diagnosis of this disease rests on the most certain signs, I dare to affirm that, by employing, with enlightened boldness, the grand method of bleeding in the treatment of acute pericarditis, results truly unlooked for will be obtained. Such, at least, is the conclusion to which the last years of my experience lead me. Almost all the cases of pericarditis which I have met, have yielded rapidly to copious bleedings, repeated several times in the space of three, four and five days. It is unnecessary to say that the bleedings, general as well as local, should be proportioned to the intensity of the disease, the age, the strength, the constitution, the sex, the complications, &c. The general rule is as follows:—in a subject in the prime of life, attacked with intense pericarditis, three or four bleedings from the arm, of three to four 'palettes,' in the three or four first days, seconded by the application of from twenty-five to thirty leeches, or by cupping, either being repeated two or three times, will suffice for the cure of the disease. I leave it to the discretion of the well-informed practitioner to determine the cases in which he ought to stop within, or to exceed the mark that we have just fixed. Few cases of pericarditis will resist this treatment, if they are taken at the commencement.

"But if, notwithstanding the assistance of art, suitably administered, the pericarditis should be prolonged, or should even pass into the strictly chronic state, it would be necessary to employ a discreet combination of moderate local bleedings, whether by means of leeches or of cupping, with various revellents, such as blisters, cauteries, moxas, setons, tartar-emetic ointment, croton oil, &c. Mercurial frictions may equally be employed.

"A severe regimen, and warm baths repeated from time to time, will favour the action of the other curative means.

"Should the effusion within the pericardium prove rebellious against all these remedies, the case becomes the most embarrassing possible. The evacuation of the liquid by a surgical operation, is one of the therapeutic problems of which experience has not yet given the solution" (Traité, i. p. 479).

Such is M. Bouillaud's treatment. It is, in fact, the pure antiphlogistic plan, (with the exception of purgatives, which he does not even name,) more actively employed than is common in France, but such as was the ordinary treatment of acute inflammations in this country for time immemorial, till the use of mercury diminished the necessity for copious depletion. In the particulars of M. Bouillaud's plan of blood-letting, there is nothing new. I do not, indeed, see any difference between his rules and those in the text above (p. 184). For robust subjects, we both recommend, as the general rule, three or four venesections and as many local bleedings, in the three or four first days; the extent of the depletion being regulated in every case, by the age, sex, constitution, intensity of the inflammation. &c. Although, however, our rules are the same in theory, they will issue very differently in practice; for while his will lead to profuse blood-letting, mine will lead to very moderate, in consequence of the action of mercury promptly suspending the indications for it.

SECTION VI.

ADHESION OF THE PERICARDIUM.*

PERICARDITIS, both acute and chronic, and especially that originating in acute rheumatism, frequently terminates in adhesion of the pericardium. Lancisi, Vieussens, Meckel, Senac, Corvisart, and more strongly than all Morgagni, are of opinion that, with a complete and intimate adhesion, the patient cannot live in a state of health. I know not how it is that Laennec and Bertin and Bouillaud have formed an opposite opinion. The former states that he had opened a great number of subjects so affected, who had never *complained* of any derangement in the circulation or respiration; whence he infers that adhesion often does not in any respect interfere with the exercise of those functions (De l'Auscult. tom. ii. p. 664). Bouillaud, in his later work, adheres, though with a slight qualification, to the same opinion (Traité, i. 447, 1835).

My own experience is entirely opposed to this doctrine. Nor has the additional experience of seven years, since the preceding sentence was written, afforded me reason to alter my opinion. I have never seen an individual with complete adhesion of the pericardium, "enjoying the most flourishing health" (Bouillaud).

The disadvantages of M. Bouillaud's plan are, 1. That profuse bleeding leaves the patient in a state of anæmic debility, from which he only slowly, and sometimes never completely recovers. 2. That the plan *fails*, if commenced late. 3. That the effusion within the pericardium is apt to "prove rebellious, when the case becomes the most embarrassing possible." 4. That valvular disease cannot be obviated with any certainty.

M. Bouillaud has, however, great merit for breaking through the trammels of habit or fashion, and resorting with boldness to the use of the lancet—a plan far superior to the imbecility of the médecine expectante. It is, perhaps, reserved for him to confer a further benefit on French practical medicine, by discarding ultra-Broussaian timidity, and exhibiting similar boldness with respect to the use of mercury. He would probably find that this remedy, discreetly employed, would save twenty lives for every one that would be sacrificed by gastro-enterite.

* This should, strictly perhaps, be ranged amongst the organic affections; but as it is in some cases more or less inflammatory long after the adhesion has taken place, and as it is intimately connected with pericarditis, it cannot be separated from it without breaking the continuity of the subject.

The *general* health, indeed, may have been flourishing, but there has invariably been more or less palpitation and hurried respiration on exertion. The absence of *complaint* on the part of the patient, Laennec's criterion, is certainly not a legitimate one; for I have often found the working classes disclaim dyspnœa even when labouring under enormous hypertrophy and dilatation, and when that symptom obviously existed in a great degree. I can only account for this by supposing that, as the symptom supervenes gradually, they get habituated to it and do not discover that it is other than their natural state. I have heard some admit, indeed, that they were "short-winded," but ascribe it to "weakness." Many others also, especially children, are naturally inattentive to their own sensations, and close interrogation is the only mode of ascertaining that, after the attack of pericarditis, they became incapable of some exercises, habits, or efforts which they previously accomplished with facility.

Further, I have never examined, after death, a case of complete adhesion of the pericardium without finding enlargement of the heart,—generally hypertrophy with dilatation. This sufficiently demonstrates the tendency of the affection. I have observed that cases of adhesion terminating in enlargement, often hurry to their fatal conclusion with more rapidity than almost any other organic affection of the heart: and I have, on the other hand, repeatedly seen patients die from the consequences of an adhesion, the history of which I could trace back eight, ten, or more years; yet such individuals would, not unfrequently, represent their health to have been perfect during the greater part of that period, and would not admit, until closely interrogated, that they had been more or less "short-winded." Hence I infer that, though close adhesion may not, for a time, create *much* inconvenience, its effects are ultimately fatal, especially in the working classes. A tranquil, abstemious life, by which, in other forms of organic diseases of the heart, existence may sometimes be prolonged to its natural period, may do much, but cannot be *equally* availing here; for, as the action of the organ itself is a constant struggle, repose is impossible.

How adhesion occasions hypertrophy is easily understood; for, first, inflammation is probably a cause of hypertrophy; and, secondly, the organ must increase its contractile energy, in order to contend against the obstacle which the adhesion, by shackling its movements, presents to the due discharge of its function; and, as explained in the article on hypertrophy, increased action leads to increase of nutrition. The cause of the co-existent dilatation is not less manifest. As the shackled organ transmits its contents with difficulty, it is constantly in a state of greater congestion than natural, and, as is more fully explained in the article on dilatation, permanent distention is the most effective cause of this affection. When the muscular substance has been softened by the previous inflammation, as frequently happens, dilatation takes place much more readily, in consequence of the deficient elasticity or tone of the heart's parietes. When valvular disease, from endocarditis complicating the pericarditis, accompanies adhesion, it will, of course, contribute its part to the production of the hypertrophy and dilatation, which must not, therefore, in such cases, be attributed to the adhesion alone.

When adhesion of the pericardium has produced hypertrophy with dilatation, its history identifies itself with that of the latter maladies, of which it renders the symptoms more severe and the progress more rapid. To avoid repetition, therefore, I refer the reader to the article on hypertrophy, and shall, here, only describe the signs which are pathognomonic of adhesion.

These signs have generally been considered very obscure. Dr. Sanders believed that he had discovered one of a positive nature in a dimple or retraction taking place, as he states, during the ventricular systole, in the epigastrium immediately below the left false ribs, and which he ascribes to the diaphragm being drawn in by the ascending movement of the heart. I have searched for this attentively in several cases of adhesion, but have not been able to detect it in any degree which could constitute a sign. Laennec, who was equally unsuccessful, thinks that it could not take place unless the stomach, by adhering both to the diaphragm and the abdominal parietes, formed the medium of retraction, for the diaphragm alone would merely draw in the false ribs.

M. Bouillaud says that "he is not yet acquainted with any sign by which we can detect adhesion of the pericardium in particular" (Traité, i. 467).

I certainly consider this diagnosis to be one of the very few connected with the heart, which cannot be made with absolute certainty, and I never, therefore, venture to *assert* respecting it; yet, in the great majority of cases, I have succeeded with much ease in detecting the affection by the following combination of signs:

1. In five or six cases (and, since this was published seven years ago, I may now say a much greater number) I have remarked one sign, which has not, to my knowledge, been hitherto noticed by others: namely, the heart, though enlarged, and when, therefore, it ought to beat preternaturally low down in the chest, beats as high up as natural, and sometimes occasions a prominence of the cartilages of the left præcordial ribs (Cases of May, Harrison, a Boy, Payne, &c.). We should, indeed, naturally expect that the adhesion would brace up the organ, and that, when enlarged and not able to descend, it must, being bounded behind by the spine, force the walls of the præcordial region forward.

2. Another sign, equally unknown to authors, and perhaps the most characteristic of all, is an abrupt, jogging, or tumbling motion of the heart, very perceptible in the præcordial region with the cylinder. It is more distinct when the heart is hypertrophous and dilated; and, under these circumstances, I have found the jogs correspond with the ventricular systole and diastole respectively, that of the diastole being sometimes nearly as strong as the other, and having the character of a receding motion suddenly arrested (Cases of May, Payne, Harrison, a Boy). This jogging motion is distinguished from the undulatory movement of fluid in the pericardium, both by its nature, by the exact synchronism of the jogs with the sounds, and by the feeling that the heart, at each systole, comes in immediate contact with the thoracic walls. I suppose the double jog to be occasioned by the heart being bound down to the spine by the adhesion, whence it is tilted forwards as often as the rounded swell of the ventricles, both during their systole (see causes of the impulse, p. 58) and their diastole, (see Experiments, p. 15,) meets the resistance of the spinal column. This view is corroborated by the same double jog having occurred in a case of aneurismal tumour behind the heart, though there was no adhesion of the pericardium and little enlargement of the heart (See *Physical Signs of Aneurism of the Aorta*). The jogs would, of course, be in-

creased by hypertrophy, and also by aortic regurgitation, which imparts abruptness to the heart's action.

3. A history of previous pericarditis, especially if connected with acute rheumatism, affords strong presumptive evidence corroborating the above signs: and the absence of such history should make the auscultator pause before he ventures on a diagnosis of adhesion in stronger terms than that "it is probable or possible."

CHAPTER II.

CARDITIS, OR INFLAMMATION OF THE MUSCULAR SUBSTANCE.

INFLAMMATION of the muscular substance of the heart may be, 1. universal; 2. partial.

1. Of universal carditis with *effusion of pus generally* throughout the muscular tissue, there is not, to my knowledge, more than a single instance on record, and that occurred to Dr. Latham. "The whole heart," says he, "was deeply tinged with dark coloured blood, and its substance softened; and here and there, upon the section of both ventricles, innumerable small points of pus oozed from among the muscular fibres. This was the result of a most rapid and acute inflammation, in which death took place after an illness of only two days."* Laennec, never having met with, or heard of, a case of this kind, and considering an effusion of pus the only unquestionable sign of carditis, says, "there does not perhaps exist a single incontestable and well-described example of *general* inflammation of the heart either acute or chronic."† Independent of the above instance, however, there are probably many others, which, though not attended with effusion of pus, will come under the denomination of universal carditis. For few will concur with this distinguished writer in excluding from the proofs of carditis, softening and induration, with increased, or diminished colour of the organ. These are results of inflammation in other muscles, and analogy points out that they may have the same origin in the heart. Further evidence is derived from the fact that, in cases of pericarditis, the characters in question sometimes occupy only a certain depth of the exterior surface of the organ; whence the presumption is almost positive that they originate in an extension of the inflam-

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* Lond. Med. Gaz. vol. iii. p. 118.

† De l'Auscult. ii. p. 554.

mation from the pericardium. The cases of this description that are on record, are too numerous to be quoted. Several have fallen under my own observation. In this point of view, then, carditis is not very rare.

As softening and induration are of sufficient importance to demand separate articles, I refer the reader to them, especially to softening, for all that remains to be said on general carditis. They are introduced amongst the organic, rather than the inflammatory affections, because authors are not entirely agreed whether they result from inflammation or from other causes, a question which I have considered in the article *Softening*.

With respect to the symptoms and treatment of carditis, they are the same as those of peri- and endo- carditis, because general carditis never exists as an independent affection, and because, when complicating the membranous inflammations, it does not present any set of signs peculiar to itself, though it greatly aggravates the general aspect of the case. I think it may be regarded as the cause of the feeble, fluttering, irregular, intermittent action of the heart, with suffocative symptoms, when these phenomena cannot be accounted for by the presence of fluid in the pericardium or of polypous concretions from endocarditis. For the symptoms and treatment, therefore, the reader is referred to Pericarditis.

2. Partial carditis, characterised by the existence of an abscess or ulceration in the walls of the heart, is not very uncommon. Bonetus, in his Sepulchretum, has described a considerable number of cases. Abscesses are more rare than ulcers. The latter occur both on the external and the internal surface of the heart, and are consequent on inflammation of the membranes of those surfaces. The external ulcer is uncommon, but Oläus Borrichius, Peyer, and Graetz have left perfect descriptions of it. The first says "Cordis exterior caro, profundè exesa, in lacinias et villos carneos putrescentes abierat." The internal ulcer is more common. Bonetus, Morgagni, and Senac present many cases. I have met with two or three.

An ulcer, whether external or internal, may perforate the heart and cause sudden death by effusion of blood into the pericardium. A perforation of the interventricular or interauricular septum may not be fatal, but will generally give rise to cyanosis. Bouillaud says that there are cases in which it does not produce any serious symptom. I have difficulty in conceiving this, and suspect that the cases were too cursorily observed.

The signs of abscesses and ulcers vary in different subjects, and are not distinguishable from those of other affections. "I know not," says Laennec "if auscultation will afford any more sure signs, and I avow that I think not." My own observation hitherto verifies Laennec's prediction. Bouillaud also says that "no positive and characteristic sign announces the existence of simple ulcerations and of abscesses of the heart. On this subject, unhappily, all pathologists are agreed" (Traité, i. 303).*

Ulceration is the most frequent cause of rupture of the heart, —fortunately a very rare occurrence. Rupture independent of ulceration generally originates in disease of the muscular tissue, by which its cohesion and resisting power are diminished:— softening for instance. I have met with one instance, mentioned in the article on dilatation. Haller and Morgagni describe many. It is generally in the left ventricle that the rupture takes place, a circumstance which at first appears remarkable, since this ventricle is the stronger; but, for the same reason, it contracts more energetically, and, as the rupture occurs during the contraction, we have thus an explanation of the phenomenon. It might be objected that supposing the strength of the muscle and the energy of its contraction to be in the direct ratio of each other, the explanation offered would not account for the phenomenon. To this it may be replied, that it is only strong muscles which *do* undergo rupture from the energy of their own contraction. Hence rupture of the auricles is much more rare than that of the ventricles.

The exciting causes of rupture are, generally, considerable efforts, paroxysms of passion, external violence—as falls, &c.

Corvisart was the first who noticed and described cases of rupture of the fleshy columns and tendinous cords of the heart: Laennec and Bertin have each met with an instance of the same. Violent efforts, as coughing, were the cause; the symptoms were, sudden and very severe suffocating dyspnœa, with overwhelming faintness, paleness and coldness, followed by all the general phenomena of disease of the heart. I have met with five or six cases of regurgitation, some through the aortic, and others through the mitral valve, which I believe to have originated in rupture or laceration of the valves, because the patients dated their malady from some violent effort, suddenly followed by the symptoms enumerated in the preceding sentence. In such of the patients as I examined after death, the suspected valve was found diseased and permanently open. In one case with similar symptoms (see case of Milton), an aneurism of the aorta had burst into the right ventricle: in another, the diseased internal and middle coats of the aorta appeared to have cracked. I should always, therefore, regard the sudden occurrence of the symptoms in question after an effort, as indicative of some serious rupture; the nature of which may, with very few exceptions, be readily ascertained by the physical signs of valvular disease explained in the chapter on that subject, and in the diagnosis of aneurisms on the pulmonary artery.

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* For partial dilatation or false consecutive aneurism of the walls of the heart, the reader is referred to the chapter on that subject amongst the organic affections.

Rupture of the heart or great vessels into the pericardium is not always immediately fatal, as a solid coagulum or a fibrinous concretion has in several instances been known to arrest the hæmorrhage for a few hours (Case by Cullerier. Journal de Med. par M. M. Corvisart, Serone et Boyer, Sept. 1806, t. xii. p. 168). Of ten cases mentioned by M. Bayle eight died instantaneously, one in about two hours, and another in fourteen (Revue Med.).

The existence of gangrene of the heart has never been distinctly proved, and the following reasons lead to the belief that its occurrence is perhaps impossible; first, the muscular tissue is one of those least susceptible of it; and secondly, inflammation of the heart sufficiently intense to occasion it, is fatal to the patient before gangrene can take place. The cases on record of reputed gangrene, appear to have been nothing more than softening, which incipient putrefaction had rendered more analogous to gangrene.

CHAPTER III.

ENDOCARDITIS, OR INFLAMMATION OF THE INTERNAL MEMBRANE OF THE HEART.

Preliminary Observations.—To M. Bouillaud the merit is due of having been the first to draw attention in a decided manner to inflammation of the internal membrane of the heart and great vessels, which had been either overlooked or only cursorily glanced at by Corvisart, Baillie, Burns, Kreisig, P. Frank, and Laennec. "In 1824 and 1826," says he, "I already possessed a sufficient number of facts to have a glimpse (entrevoir) of all the importance of inflammation of the heart and great vessels. But those facts did not yet permit me to treat this rich and fertile subject with all suitable minuteness: hence, the ideas which I broached were regarded by many physicians of great authority as purely theoretical" (Traité, ii. p. 1, 1835). This neglect may be attributed mainly, I think, to the manner in which M. Bouillaud treated his subject: for he concentrated his attention principally on aortitis—a disease so obscure that it might well be supposed theoretical, while he almost overlooked the immense importance of endocarditis, and certainly failed to throw any correct light on its symptoms. For instance, he devotes three pages to the signs of aortitis, and dismisses endocarditis with the following passage: "*As to independent inflammation of the membrane of the heart, it is excessively rare:** Analogy indicates that it ought to be characterised by augmentation of the force and frequency of the beats of the heart, when it is not sufficiently violent to diminish, suspend or entirely arrest the action of that organ. Observation confirms that which analogy leads us to foresee. In fevers properly so called, *which appear to me to be invariably accompanied*

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* M. Laennec had said, "Inflammation of the internal membrane of the heart and great vessels is a very (fort) rare affection:" thus he follows Laennec with respect to endocarditis.

with irritation of the heart, the frequency and force of the pulse are the two principal phenomena which strike our attention: if the fever assumes a grave character, and occasions so serious (profonde) an irritation of the heart, that the muscular substance itself of the organ becomes affected, the pulse loses in force and regularity, while it increases in frequency, and an unexpected death frequently terminates this always formidable complication" (Traité du Cœur par M. M. Bertin et Bouillaud, p. 69, 1824). As this doctrine of fever was not considered sound, it increased the belief that M. Bouillaud was describing an imaginary disease.

Another reason why he excited opposition to his own views, was, that he ascribed, *with too little qualification*, all varieties of redness and all kinds of depositions in the heart and aorta to inflammation. In his recent work he disavows having done so, and repels the charge as a misapprehension of others; for, says he, "I had declared in the most express manner, that, amongst those varieties of redness, there were some purely cadaveric or from imbibition of blood after death" (tom. ii. p. 2). This declaration, however, I have not been able to find in the Traité du Cœur of M. M. Bertin and Bouillaud in 1824; nor is there, in that work, any reference to a *Treatise on Fevers*, cited in his later Treatise on the Heart in 1835. After describing, in the Traité in 1824, all the varieties of redness, including one which he compares to a *stain*, (and which every one now admits to be nothing else,) he finally says, "We have considered the redness to be the result of inflammation, *whatever was its shade.*" The utmost qualification of this opinion that I find, is, "If any shade of redness can, in fact, be regarded as not inflammatory, it is beyond contradiction that in question (viz. violet). New facts are necessary to enable us to pronounce in a positive manner on its true character . . . . In conclusion, the violet, brown and even blackish colour, is not a decisive reason for rejecting the idea of inflammation; for many inflammations of the skin and mucous membranes, and, amongst others, those which manifest a gangrenous tendency, are accompanied with livid, violet, brown or blackish redness" (Traité, par M. M. Bertin et Bouillaud, p. 55 and 56). Now, surely, this is almost the opposite of a "declaration in the most express manner, that there were some (varieties of redness) purely cadaveric or from imbibition of blood after death."

With respect to depositions, he says, "The yellow points, the cartilaginous patches, the calcareous and plaster-like incrustations of the aorta, appear to us to be nothing more than a series of metamorphoses successively passed through by the matter secreted by inflammation" (p. 58). Such being M. Bouillaud's opinions in his own words, he must necessarily be under some mistake in disavowing them and imputing misapprehension to others.

I was not one of those who rejected the opinions of M. Bouillaud as "theoretical," though, as will presently be shown, he erroneously states that I was. I adopted his account of redness as far as he *now* (1835) admits it himself; but I pursued the train of reasoning followed by Laennec (because no better was necessary) to prove that redness was sometimes a result of sanguineous imbibition, and to show how the latter might be discriminated from the inflammatory kind. I have not seen cause to alter this train of reasoning in the present edition. Further, I gave a full account of the anatomical changes indicating what I considered to be, really, inflammation of the interior of the heart and aorta (1st edit. p. 148): again, amongst the exciting causes of diseases of the valves, I specified "*inflammation of the internal membrane of the heart, resulting from carditis, pericarditis*—especially rheumatic, from fever or from any other cause" (p. 319): lastly, I discovered and published the grand pathognomonic signs of acute endocarditis, namely, the valvular murmurs, at a time when they were not only unpublished, but possibly unsuspected by M. Bouillaud; for he states (Traité, ii. p. 2) that he conducted his researches on acute endocarditis especially, during the years 1832-3 and 4, when my work, published in Dec. 1831, (though dated by the publisher 1832,) was accessible to him. It cannot be said, therefore, that I had not, in 1831, both recognised, corrected and extended the very limited and imperfect researches of M. Bouillaud on endocarditis. During the ensuing three years, I had so far widened my observation, that, when his work appeared in 1835, I can frankly say that the article endocarditis scarcely contained a material fact to which I was a stranger.*

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* The above remarks evince, I will not say the *injustice*, (because, as M. Bouillaud is said to be unacquainted with the English language, he may only have been mistaken,) but the incautious *inaccuracy* of the following observations in his last

SECTION I.

ANATOMICAL CHARACTERS OF ACUTE ENDOCARDITIS.

The anatomical characters of acute endocarditis are, redness of the internal membrane of the heart and arteries, an effusion of lymph or pus on its surface, and thickening, softening and ulceration of its substance and of the subjacent cellular and fibrous tissues; also, according to M. Bouillaud, the presence of adherent, colourless coagula of blood. Each of these characters will be considered in succession.

A. *Redness of the internal membrane of the heart and arteries.*—This is sometimes inflammatory, and sometimes not. We will first notice the latter.

1. Redness not inflammatory, often appears in the aorta, the pulmonary artery, and the heart, and is a *uniform*, intense colour, as if stained by the blood. Corvisart (p. 36) avows that he cannot give a satisfactory account of its nature and cause. P. Frank

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Treatise. "In the chapter devoted to inflammation of the interior of the heart and arteries, Dr. Hope *has scarcely added anything to what had already been published on the subject by me in* 1824, *and he has thought proper to make himself, in some sort, the echo of all that M. Laennec had said against the intervention of appreciable inflammatory action*, which I had admitted as the point of departure of a certain number of lesions, called *organic*, of the valves of the heart or of the walls of the aorta. Further, like Laennec, Dr. Hope *teaches us absolutely nothing, either on the causes or on the diagnosis of inflammation of the internal membrane of the heart*" (vol. ii. p. 6).

He proceeds, "It is seen from what precedes to what a *state of penury*, if I dare so express myself, medicine was reduced on the important point which occupies our attention. The new facts which I have collected during the last three years, (1832, 1833 and 1834,) permit me, however, to affirm that inflammation of the internal membrane of the heart is, contrary to the opinion of M. Laennec, a disease really very common, and as frequent as pericarditis itself."

It is but justice to my countrymen to say that not only this fact, but almost every other of importance which M. Bouillaud has published either on endocarditis or pericarditis, is to be found in the works of Dr. Latham, Dr. Elliotson, Dr. Stokes and myself, all published not only before M. Bouillaud's Treatise, but, with one exception, (Dr. Stokes in 1832,) before he had even commenced his researches on endocarditis in 1832. I may add that he is still singularly in the rear in his diagnosis of endocarditic valvular affections; since he does not even pretend to specify the *particular* valve diseased, the mode of accomplishing which I had distinctly pointed out in 1831, but which is brought to the utmost nicety in the present edition.

regarded it as an inflammation of the arteries, which, according to him, occasioned a peculiar and almost always fatal fever (De Curand. Homin. Morbis, tom. ii. p. 173). Bertin and Bouillaud "have considered it, whatever was its shade, as the result of inflammation" (Traité, p. 55). Laennec entertains an opposite opinion, and demonstrates satisfactorily that the redness in question, *when not accompanied by other anatomical characters of inflammation*, is the result of sanguineous imbibition.* As it is necessary that the reader be able to judge for himself, I shall give some account of this redness, adhering to the description of Laennec, which I have verified by repeated experiments and dissections.

The redness is sometimes scarlet, and sometimes brown or violet.

a. The scarlet redness of the interior of the arteries is often confined to the internal membrane exclusively; and, when that membrane is removed by scraping with the scalpel, the subjacent cellular tissue and the fibrous coat are found as pale as in their natural state. But in other cases the redness penetrates more or less deeply into the fibrous coat, and sometimes it reaches, in parts, even the cellular or external tunic. The redness of the internal coat is a perfectly uniform tint, similar to that which would be presented by a piece of parchment painted red. No trace of injected capillaries can be distinguished in it; but the tint is sometimes deeper in one part than another. Sometimes it diminishes insensibly from the origin of the aorta to the place where the redness ceases: but, very often, it terminates suddenly, forming abrupt borders of an irregular shape. Sometimes, in the midst of an intensely red portion, is found an accurately circumscribed patch of white, which produces precisely the effect that is occasioned by an impression of the finger on a part of the skin affected with phlegmon or erysipelas. When the aorta contains very little blood, the redness only exists in the tract in contact with it, and forms a sort of ribbon. The origin and arch of the aorta are the parts of that artery which are the most frequently found thus reddened. Sometimes nearly all the arteries present the stain. The aortic and mitral valves participate in it, and appear as if they had been immersed in a red dye. Though the red is scarlet in the arteries, it is deeper on the valves, approximating slightly to purple or violet. This proceeds merely from deficiency of the opake white ground, which enriches the colour in the aorta by reflecting light.

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* It might be added, *or when not preceded by distinct physical and general signs of acute endocarditis*; for these signs, when distinct, are so unequivocal that I agree with M. Bouillaud in thinking them sufficient to prove the redness inflammatory, provided the subject be not opened later than twenty-four hours after death; as, after this period, the colour may result from putrefactive imbibition.

When the pulmonary artery is reddened, its valves and the tricuspid are also very commonly stained in the same way. The stain of the right cavities and vessels of the heart is always of a deeper and browner hue than that of the left—a circumstance dependent, in all probability, on the darker colour of the venous blood. The internal membrane, where it invests the muscular substance of the ventricles and auricles, sometimes does not present any sensible change of colour, even when the valves are vividly reddened. More commonly, however, it participates in the redness, but exhibits a darker, and more violet or browner hue, simply in consequence of the ground being deeper.

The redness described is not accompanied with any sensible thickening or vascular injection of the stained membranes. It is not removed by washing, but a few hours' maceration in water suffices to make it totally disappear.

Such are the characters of the scarlet redness. We next come to—

b. The brownish or violet stain. It is found equally in the aorta, the pulmonary artery, the valves, the auricles and the ventricles. Most commonly, indeed, it is observed in all these parts simultaneously. It is often very unequal in intensity, and is always deeper on the parts of the vessels which, according to the laws of gravity, have been most in contact with the blood. Its shade is, of course, less deep on the valves and in the arteries than over the muscular substance, because this forms a darker ground. It is not so commonly restricted to the lining membrane as the scarlet redness; for the muscular substance of the auricles and ventricles, and even the fibrous tunic of the aorta and pulmonary artery, usually participate in the dye,—at least in some points and to a certain depth.

Such is Laennec's account of redness of the internal membrane. But redness, he contends with great justice, is not sufficient to characterize inflammation, particularly when it is not accompanied by thickening or vascular injection of the reddened parts. Moreover, the abrupt circumscription of the redness, in some cases, within geometrical, though irregular lines, (an appearance never seen in inflammation of serous membranes, though it presents itself occasionally and to a slight degree in that of mucous,) banishes the idea of inflammation, and conveys that of a stain by a coloured liquid, which had run irregularly on the reddened membrane, and, on account of its deficient quantity, had not been able to touch every part.

Again, the circumstances under which the redness is usually found, countenance the idea of its being a stain, rather than from inflammation. Thus, Laennec found the scarlet red to occur after a somewhat protracted agony in subjects still vigorous, but cachectic in consequence of disease of the heart or some other malady. The blood in these cases was never very firmly coagulated, and the body most frequently presented some signs of decomposition.

The brownish or violet red, he found in those subjects especially, who had died of continued typhoid fevers, of emphysema of the lungs, or of diseases of the heart. Almost all had experienced a long and suffocative agony; in all, the blood was very liquid and evidently altered, and signs of premature decomposition existed in the bodies. I have myself also very constantly found it in cachectic subjects affected with passive hæmorrhage from the gums, from ulcers, or from any tender or broken surfaces,—as in scurvy and purpura. It is, moreover, in summer particularly, and in subjects that are opened more than twenty-four hours after death, that the dark discoloration is most frequently met with.

Both varieties of redness, and particularly the brownish or violet, are accompanied with a greater or less degree of softening of the heart, and with an increased humidity of the arterial walls. In most instances, these states are evidently the effects of a commencement of putrefaction.

The cases which Bertin and Bouillaud have adduced in substantiation of their opinion that the redness in question is of an inflammatory nature, are strikingly corroborative of the opposite views of Laennec. For, of twenty-four cases, eleven are typhoid fever, or other affections, in which there was a manifest alteration of the liquids, and premature putrefaction. The thirteen other cases consist almost entirely of consumptive patients; and the writers observe, in general terms, that the redness appeared to coincide with a remarkably fluid state of the blood. It must, further, be remarked that most of their examinations were made in summer, and more than thirty hours after death.

In order to ascertain experimentally whether blood could occasion a stain, Laennec enclosed a quantity in a sound and recent aorta, and placed the preparation in the stomach of the subjects, in order to preserve it from drying, and to put it under the same circumstances of decomposition as the rest of the body. In twenty-four hours it presented a perfect specimen of the scarlet dye, which was not weakened by reiterated washing.

He found that blood, too firmly coagulated, causes imbibition feebly and slowly: that blood half coagulated, and particularly the blood, still slightly florid, which may be pressed out of the lungs, produces the scarlet redness: that very liquid blood, and particularly that with a serous intermixture, produces a violet colour of greater or less depth: and that if the artery be only partly filled, the dye occupies those parts alone which are in contact with the blood, thus forming a ribbon. If the walls of the artery are firm and elastic, the dye, he continues, requires a long time (seventy or eighty hours) for its formation, and is never very deep; but if, on the contrary, the walls are soft, supple, and charged with humidity, the dye promptly penetrates through the whole thickness. Warm weather and the rapid progress of putrefaction are favourable to the imbibition.

Boerhaave and Morgagni also attributed the red colour to the stagnation of blood which takes place during the agony in diseases accompanied with great oppression; and Hodgson likewise maintains that arterial redness, such as that above described, does not arise from acute inflammation, as it is not accompanied by any other anatomical characters of inflammation. When occurring in the vicinity of coagula, it is, he thinks, an effect of imbibition after death.

It is impossible not to conclude from all the evidence now adduced, *first*, that redness of the internal membranes of the heart and arteries cannot *alone* prove inflammation; *secondly*, that it is a phenomenon taking place during the agony, or after death, whenever it is found in conjunction with the following circumstances: namely, a prolonged and suffocative agony; manifest alteration of the blood; and a somewhat advanced decomposition of the body.*

Such is the redness of the internal membrane of the heart and arteries which is not inflammatory. We now proceed to that which is.

2. The colour of inflammatory redness may be the same; for the membrane, though inflamed, is still liable to imbibition. In the absence of imbibition, the redness is fainter, less shining, more equably diffused, and less characterized by streaks, patches, isolated unstained spots, and abrupt edges. The further proofs that it is inflammatory, fall under the next head. The absence of all redness does not exclude the idea of inflammation; for, in other serous membranes, when slight, it sometimes disappears after death.

B. *Effusion of lymph on the internal membrane, with thickening of its substance.*—Whether redness be due to vascularity alone, or to this, conjoined with imbibition, its inflammatory nature is known by the presence of other anatomical characters of inflammation. These are, thickening, swelling, and puffiness of the inner membrane, especially about the valves; an effusion of lymph on either its free or adherent surface; and a preternatural vascularity, with softening and thickening, of the middle arterial coat. Each of the coats, also, may be separated from the other with much greater facility than natural, by scraping with the nail or scalpel, in consequence of softening of the interposed cellular tissue. The internal and middle coats and their connecting cellular tissue, in short, present all the phenomena of the adhesive inflammation as it displays itself in other membranes. It is by this inflammation that, if an artery be wounded or divided; if it be compressed by a ligature or tumor; or if it be simply irritated by ulceration of the surrounding parts or a pulmonary vomica, an effusion of lymph takes place into the cavity of the vessel and into the cellular tissue, both investing it externally and connecting its several coats together, by which the caliber of the vessel is obliterated and hæmorrhage prevented.

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* Though M. Bouillaud has denounced me as the echo of Laennec against inflammation, he has himself come round to my views, and reproduced, almost totidem verbis, the conclusions in the text. The following are his words—

First, "I do not think it possible to decide by simple inspection, nor by washing or maceration itself, whether a given redness of the internal membrane of the heart be the effect of inflammation or of cadaveric imbibition. It is necessary therefore to search elsewhere for the means of resolving this important question."

Secondly, "I have convinced myself by a great number of facts, that certain varieties of redness of the heart and vessels are nothing more than a purely cadaveric imbibition; and I have ascertained, in common with a great number of other observers, that these latter varieties of redness exist almost constantly in individuals opened at a time when putrefaction of the body is already more or less advanced, especially if those individuals have died of a disease which has been accompanied by putrid or typhoid phenomena—in which case the blood is more liquid than in the normal state, a circumstance that renders it more susceptible of imbibition by the internal membrane of the vascular system.

Lymph has been found effused on the unattached surface of the lining membrane within the auricles and on the valves, by Baillie,* Laennec,† and Burns.‡ I have met with it both in the heart and aorta.§ Effusions of lymph within the heart and great arteries, however, are very seldom *found*; and hence it is, that Laennec thinks inflammation of the internal membrane of those parts extremely rare. (De l'Auscult. ii. p. 498). But the presence or absence of lymph is not sufficient to determine whether inflammation exists or not; for, in many instances, the lymph, when first effused, is unquestionably washed away by the force of a circulation so powerful as that in the heart and aorta. The same remark applies still more strongly to pus. It is, however, as Bouillaud well remarks, "sometimes concealed in the centre of coagula, or detained in the meshes formed by the columnæ carneæ."

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* Morbid Anat. Edit. 5, p. 85.

† De l'Auscult. tom. ii. p. 127.

‡ On Diseases of the Heart, chap. 9.

§ The three preceding paragraphs demonstrate the inaccuracy of M. Bouillaud's representation, that I had denied the intervention of inflammation as a cause of redness and morbid organic changes. Subjoined are his own criteria, the anatomical parts of which are identical with those in the text: the semeiological part (viz. endocarditic murmurs, &c.) I had published before himself, as already shown at p. 202.

"In my opinion," says M. Bouillaud, "we may consider as of an inflammatory nature a redness of the internal membrane of the heart, existing in an individual whose body has been opened before any trace of decomposition had shown itself, and which had presented during life the symptoms that we shall assign in the following article to inflammation of the internal membrane of the heart. But the inflammatory nature of the redness will be still more evident if, to the circumstances just specified, the following conditions be united: 1. swelling, thickening, and tumefaction of the parts occupied by the redness; 2. the presence of a certain quantity of pus, of false-membranous matter, or even of those adherent, colourless coagula, which resemble the inflammatory buff of the blood or fibro-albuminous lumps (pelotons); 3. the co-existence of similar redness in blood-vessels, the inflammation of which was positively ascertained before the death of the patient" (Traité, ii. p. 173, 1835).

Kreysig, Hodgson, Bertin and Bouillaud, and Bouillaud in his later work, are of opinion that lymph effused by inflammation is the source of fungous or warty vegetations of the valves. Laennec rejects this opinion, and attributes the vegetations to sanguineous concretions, which adhere to the internal membrane and become organised. He does not deny, however, that an inflammatory false membrane may become the nucleus of these concretions. I once caused the deposition of granulations in an hour, by lacerating with a hook the pulmonic valves and interior of the right ventricle of an ass poisoned with woorara (see p. 39); which, I think, militates in favour of the inflammatory origin of vegetations, as the general rule; but it is probable that the cause assigned by Laennec is occasionally real. This subject will be more fully considered under the head of Vegetations.

C. *Ulceration of the Internal Membrane.*—Ulceration of the internal membrane sometimes takes place from acute inflammation, and it may exist without occasioning any lesion of the subjacent tissues. One instance is given in Case 50 of Bouillaud (ii. p. 48). I think, however, that acute ulceration is rare; for, in general, ulceration is manifestly a consequence of some previous chronic degeneration of the coats of the vessel, and is, in the first instance, rather a solution of continuity than an ulceration. Such is the case when it is occasioned by the detachment of calcareous incrustations, or by the deposition of atheromatous or other matter underneath the internal membrane. As these depositions are rare under that part of the membrane which covers the muscular substance, we see the reason why ulcers are seldom found within the cavities of the heart. Still, I have seen four or five instances of this in a chronic form.

I have never seen or heard of a case in which endocarditis manifestly terminated in gangrene. Bouillaud, however, has collected four or five cases in which "he is tempted to think that the very rapidly fatal termination was attributable to an endocarditis strongly analogous to certain gangrenous inflammations" (See tom. ii. p. 176, and Cases 22 and 39). These cases, though open for consideration, are far from being conclusive, as a previously diseased state of the blood would sufficiently account for the symptoms which they presented.

Coagulation of the blood within the heart, as a consequence of acute endocarditis, is a phenomenon of which I have no personal knowledge, because I have never witnessed a fatal case of this affection: but I entertain no doubt of its reality; first, because such coagulation is a well-known result of phlebitis and arteritis; and, secondly, because M. Bouillaud, who has been more fortunate in seeing fatal cases, gives the following account of the coagulation:—

"It results from the numerous cases of acute endocarditis given in my first category, that this inflammation commonly induces, as its consequence, the coagulation of a greater or less quantity of the blood which circulates through the cavities of the heart. In this respect endocarditis resembles arteritis and phlebitis. The concretions of blood formed by acute endocarditis must not be confounded with the ordinary clots met with in the heart, especially those formed after death. The concretions consequent upon acute endocarditis are white, colourless, elastic, glutinous, adherent to the walls of the heart, twisted round the valvular tendons and fleshy columns. They are in a manner half organised, and, as I have already said, strongly analogous to the inflammatory buff of the blood, or to false membranes themselves: some occasionally present red points and lines, which are really nothing more than rudiments of vessels.

"The concretions in question differ much in volume and configuration. They extend pretty commonly into the great vessels. They are, cæteris paribus, larger and more abundant in the right cavities than in the left. Their most adherent part is generally about the free border of the valves, where some fragments may still remain after reiterated washing. It is probable that these little fibrinous masses may become organised or transformed into vegetations or granulations" (Traité, ii. p. 178).

Such are the anatomical characters of acute endocarditis. It is scarcely necessary to add, that, if resolution and *complete* absorption do not take place, the thickening of the lining membrane becomes permanent. Lymph adhering to its surface becomes organised. Laennec thinks, as already stated, that adherent coagula of blood occasionally undergo the same change. Lymph deposited beneath the lining membrane, also becomes organised. Though the valves are its principal seat, it does occur, and occasionally in a very marked degree, under the membrane investing the muscular substance; for I lately saw an instance in which several masses, as large as peas and horse-beans, existed under the membrane of the left ventricle. In slighter cases, we see this membrane present a mottled opacity from subjacent thickening. The whole of these accidental depositions, especially those connected with the valves, may, according to the laws of Analogous Transformations, pass into the successive states of fibrous tissue, cartilage and bone. For all the details on this subject, the reader is referred to the section on the Anatomical Characters of Diseases of the Valves. It may be finally stated that it is these ultimate organic changes, constituting incurable and often fatal disease of the heart, which render endocarditis one of the most important and formidable diseases in the nosology.

SECTION II.

SIGNS AND DIAGNOSIS OF ENDOCARDITIS.

This subject need not detain us long, as most of the signs are analogous to those of pericarditis, which, in the great majority of cases, complicates endocarditis. I therefore thought it desirable to include a pretty complete notice of endocarditis in the chapter on Pericarditis, because, as the latter name is familiar to practitioners, it will continue to be turned to for a considerable period before the new, but appropriate term *endocarditis*, which owes its origin to M. Bouillaud, becomes universally known.

It has appeared to me that endocarditis more frequently exists without pericarditis, than this without the other. M. Bouillaud gives 34 cases of endocarditis, of which one half were exempt from pericarditis. I shall assume, in the following account, that the endocarditis is uncomplicated.

General Signs.—Inflammatory fever exists in a greater or less degree, but its symptoms are suspended when great embarrassment of the circulation supervenes, and are replaced, as will presently be shown, by the symptoms of apnæa.

Pain is represented by Bouillaud to be entirely absent, except when it is attributable to coexistent pericarditis or pleuritis. I think that this is rather overstated, as I have several times noticed a slight pain in apparently pure endocarditis. Though there be no pain, there is always an undefinable "*uneasiness*" in the præcordial region, often attended, I have observed, with a somewhat anxious, distracted expression of countenance. It will presently be shown that this uneasiness amounts to insupportable distress when the circulation through the heart becomes greatly impeded.

Here the symptoms (as in pericarditis) branch off into two widely different classes, according, 1. as the circulation through the heart continues free; 2. as it becomes greatly obstructed by valvular disease or polypi.

1. When the circulation continues free, the action of the heart, stimulated by the inflammatory irritation, is violent and abrupt. The increased extent over which it is perceptible is, I think, proportionate to this violence, rather than to inflammatory turgescence of the organ, as supposed by M. Bouillaud. I have known a tremour, about the 4th and 5th left intercostal spaces, accompany the impulse when there was regurgitation through the mitral valve.

The pulse, corresponding with the action of the heart, is, as a general rule, full, strong, hard and regular, but there are occasional exceptions in weakly, nervous subjects. Aortic regurgitation renders it jerking, and sometimes imparts a thrill to the arteries when the circulation is strong. Its frequency I have found to range principally between 80 and 110.

Respiration is slightly accelerated, as in other inflammatory affections; but, while the patient is at rest, there is little or no oppression.

The circulation being free, there is no purpleness or puffiness of the face, or œdema with coldness of the limbs, indicating venous retardation; nor any wandering of the mind, from the circulation of venous blood through the brain.

This series of symptoms constitutes a very supportable form of disease,—more so even than pericarditis without effusion of serum; for in the latter there is often pain and, consequently, a constrained position on the back. Still, it must never be forgotten that, mild as is this form of endocarditis, it is equally dangerous in its ultimate results,—valvular disease, as the most distressing form next to be described.

2. When the circulation through the heart becomes greatly impeded, whether from the orifices being obstructed by a tumid and contracted state of the valves, or from their admitting of free regurgitation, or from the blood coagulating and choking up the cavities of the heart, or entangling and impeding the action of the valves, another class of symptoms, of the most distressing kind, presents itself—a class analogous to that produced in pericarditis by much fluid in the pericardium compressing the heart, and which was pointed out by the writer as also characteristic of polypus, independent of endocarditis, when formed before death (see Signs of Polypus, or p. 511 of the 1st edit.).* I believe, however, that when cases of acute rheumatism and of inflammation of the heart are treated in the way that I have pointed out, (p. 179 and p. 184,) the severe symptoms in question will be of very rare occurrence:—at least, I have never seen them in a considerable degree. I therefore admit them principally on the authority of M. Bouillaud. They are as follows.

The action of the heart becomes irregular, unequal, intermittent, and exceedingly quick, attaining from 130 to 160 or more beats in a minute. Sometimes, beats are dropped in the pulse which exist in the heart, every contraction of the organ not expelling blood enough to propagate an undulation into the arteries.† The impulse is sometimes simultaneously violent, from the struggle of the heart against the obstacle; but it ultimately becomes feeble and fluttering from exhaustion.

The pulse is generally small, weak, irregular, unequal and intermittent, and this may be the case though the impulse be violent and tumultuous; for, says M. Bouillaud, the large fibrinous concretions in the ventricles, and the obstructions in the valves, are circumstances which, in spite of the violence of the heart's contractions, prevent the projection of a large column of blood into the arteries. When there is free aortic regurgitation, the pulse will, of course, be jerking.

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* The same symptoms are produced by paralysis of the heart from poisons: they therefore indicate any *extreme* impediment to the circulation through the heart, whatever be its cause. This analogy is interesting and instructive.

† M. Bouillaud introduces this as "a new species of *désaccord.*" It was fully described in the first edit. of this work, p. 331-2.

From this defect of arterial circulation result ghastly paleness, coldness, mortal faintness and actual syncope, overwhelming anxiety of mind and countenance, perpetual jactitation, and an agonizing feeling of suffocation which confines the patient to the erect position, and prevents the possibility of a moment's sleep.

The venous circulation being retarded, any redness of the face and hands becomes purple or livid, and, if the patient survive a few days, dropsy may show itself in puffy intumescence of the face and œdema of the lower extremities. The mind, too, may wander a little, from the circulation of venous blood through the brain; and, occasionally, when this organ becomes much congested, there may be sudden insensibility, slight convulsive movements, stertorous respiration and foaming at the mouth. These symptoms occurred in two cases recorded by Bouillaud (see tom. ii. p. 208).

The class of severe symptoms now described very rarely exists in a marked degree dissociated from the mechanical causes to which they are ascribed. Yet, in a few cases, I have known weakness, irregularity, intermission and inequality of the beats of the heart, together with orthopnœa, anxiety and distress, to exist *temporarily and in a moderate degree*, though the general context of the cases led me to judge that there was no great mechanical impediment to the circulation though the heart. The same may occur in pericarditis without serous effusion. Here then we see the intervention of a disturbed state of the nervous system, and we must no more overlook its occasional and possible influence in these cases, than when the symptoms in question result, as they often do, from a mere fit of dyspepsia, bile, gout or hysteria.

*Physical Signs.**—*Percussion* is dull over a surface of 4, 9, and even 16 square inches. M. Bouillaud, if I understand him, ascribes this to "turgescence of the heart, from the inflammatory fluxion" (tom. ii. p. 205).

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* M. Bouillaud says, these "have not yet been noticed by any author" (ii.

I cannot easily comprehend how the walls of the heart can simply *swell* to such an extent; but I can readily conceive that the effect might be produced by distension of its interior by polypi or blood; for, in experiments on rabbits poisoned with woorara, I have seen the heart swell to nearly double its natural size from engorgement, when artificial respiration was temporarily suspended (see p. 23). I am disposed to think, therefore, that increased dulness on percussion will be slight or absent in cases of endocarditis where the circulation is free, and exist in a high degree in those only, in whom there is a great impediment to the circulation, attended with the second, or distressing class of general signs above described.

Dulness from this cause may be discriminated from that produced by fluid in the pericardium by the impulse (when not imperceptible from feebleness) *sensibly* striking the walls of the chest, and by its being exactly synchronous with the first sound; whereas in hydropericardium it is indistinct, undulatory, and not synchronous. Further, the dulness of much fluid in the pericardium mounts higher up the sternum than that from endocarditis.

Impulse.—This is violent, abrupt and regular, so long as the circulation through the heart is free. When it is greatly impeded, as indicated by the weak, irregular pulse, the impulse may for a time continue violent, but it is an irregular, confused tumult; and this violence generally subsides into a feeble, unequal flutter as the obstruction increases and the nervous power fails.

Sounds.—If the inflammation has caused constriction of either set of sigmoid valves, or permanent patency of either auricular valve allowing regurgitation, a murmur will attend the *first* sound, and it may proceed either from the sigmoid valves alone, the auricular alone, or from both conjointly.

If the inflammation has caused permanent patency of either set of sigmoid valves, with regurgitation, a murmur will attend

204). He is mistaken; he forgets that he himself commented, in his previous volume (p. 200), on my account of the valvular murmurs of endocarditis, where he also alludes to the labours of Dr. Stokes and Dr. Latham.

the *second* sound. I think that it seldom if ever proceeds from contraction of the auricular valves.

In the vast majority of cases, the murmurs are confined to the *left* side of the heart. It is obvious that if polypi should almost choke up the passage through the heart, the murmurs would diminish or wholly cease, as there would not be a sufficient current of blood to produce them.

For the mode of easily ascertaining which is the *particular* valve affected, the reader is referred to *Valvular Disease, Physical Signs;* and, for the *Diagnosis* of valvular, from attrition-murmurs occasioned by pericarditis, he is referred back to p. 174.

Summary.—Such are the general and physical signs of endocarditis, and I may now sum up by stating that this affection may be anticipated if a person be *suddenly* attacked with three signs: 1. Fever; 2. Violent action of the heart; 3. A valvular murmur which did not previously exist, provided the murmur be well distinguished from an attrition-murmur, as the latter indicates pericarditis. The evidence is still stronger if the signs occur in connexion with acute rheumatism.

SECTION III.

CAUSES, PROGRESS AND DURATION, TERMINATIONS, PROGNOSIS AND TREATMENT OF ENDOCARDITIS.

THE *Causes* of endocarditis are the same as those of pericarditis, (see p. 176) to which phlebitis, extending to the heart, may be added.

Progress, Duration and Terminations.—If the treatment of endocarditis be commenced early and conducted vigorously, and especially if acute rheumatism—its ordinary cause, has been treated on the principles explained in the preceding chapter, (p. 179, note,) the disease may, according to my experience, generally be divested of all danger to life in three or four days or a week: M. Bouillaud calculates eight days to be about the average term; but he does not employ mercury. I feel satisfied that *complete* cures, without a trace of murmur from valvular disease remaining, may be effected in a considerable proportion of cases within the term which I have specified --and more readily, indeed, within this period than afterwards; for I have observed, that when a murmur continues more than a week or ten days, it is apt to resist for several weeks longer, and sometimes permanently: which, indeed, is what we should expect; for when the morbid products of inflammation have once become organised, (and this process may commence in less than a week,) they are far more difficult to remove.

If the murmur should persist beyond a week or ten days, the endocarditis may be regarded as passing into the chronic stage, and this may continue for several weeks or even months, and still be benefited by antiphlogistic treatment. After this, if the murmur be not subdued, the affection enters into the list of established valvular diseases; which, if neglected, may ultimately compromise life, but if suitably treated, may, as will be shown in the Section on the Treatment of Valvular Diseases, be prevented, in a large proportion of cases, from producing serious consequences.

The termination of endocarditis in valvular disease has, I fear, been by far the most common up to the present time, especially amongst the working classes. This is in consequence of endocarditis having been little known as an effect of acute rheumatism; whence the treatment of the latter was not specifically directed to the obviation or removal of the former. In proof of this, I may repeat a statement already made, that I have found the worst forms of valvular disease to date more frequently from "rheumatic fever," (by which is to be understood rheumatic endocarditis,) than from all other causes put together. The eyes of the profession are now attentively directed to this subject; and it is to be hoped that it will soon become one of the best known, because most important, in medical science—one, in short, of which it will be disgraceful to be ignorant.

The *Prognosis* of endocarditis may be collected from what has now been said. The acute affection, with good diagnosis and treatment, is rarely fatal. But, according to M. Bouillaud, it *may* be fatal "in the space of a few days, and then, most commonly, one of the principal causes of death is the formation of concretions of blood in the cavities of the heart" (Traité, ii. p. 282). Chronic endocarditis presents a gloomy remote prognosis, in consequence of the probability of confirmed valvular disease.

The *Treatment* suitable for acute endocarditis is the same as that for pericarditis, (see p. 184,) and *it must not be less prompt and vigorous.* The practitioner must not be misled by the apparent mildness of the symptoms in cases where there is little impediment to the circulation through the heart. He must never, for an instant, forget, that there is a *possibility* of subsequent valvular disease, and that the mere possibility is a contingency of such magnitude, as to merit all the resources of his abilities and experience for its obviation.

In chronic endocarditis, I have experienced the most satisfactory results from prolonging the mild use of mercury, so as to maintain a barely sensible effect on the gums, for three, four, five, or six weeks; simultaneously employing a succession of small blisters on different parts of the præcordial region, restricting the patient to a farinaceous and light broth diet, and confining him to bed, for the purpose of *ensuring* the utmost possible corporeal tranquillity.

Should the murmur still resist, the mercury may be discontinued, and its future resumption must be left to the judgment of the practitioner; but the counter-irritant, antiphlogistic treatment, in a moderate degree—that is, short of reducing the patient to a state of anæmic debility, together with quiet and the use of digitalis and mild sedatives, as extr. hyoscyami and tr. or infus. lupuli, may be advantageously continued for several months, with the view of completely subduing the chronic inflammatory process, and allowing any thickening that has already taken place, to undergo the utmost possible absorption. Beyond this, the treatment resolves itself into that of established valvular disease, for which the reader is referred to the chapter on that subject.

CHAPTER IV.

ACUTE AND CHRONIC ARTERITIS, AND ORGANIC DISEASES OF THE COATS OF ARTERIES.

ACUTE ARTERITIS. — The anatomical characters of acute arteritis are the same as those of acute endocarditis, described at p. 203, but they are much more difficult to ascertain, because lymph, pus, and coagula are seldom found in the aorta, and the tumefaction of its coats is not so easily appreciated as that of the valves. It is still more difficult, if not totally impossible, to assign to acute arteritis any particular set of symptoms, because it is perhaps always complicated with endocarditis, by which its own symptoms are, as it were, absorbed: to speak more explicitly, the two diseases are essentially one and indivisible. Hence it was, that, before endocarditis was thoroughly understood, the symptoms common to both were, by Bertin and Bouillaud, ascribed principally to aortitis. In the first edition of this work, I pointed out the numerous fallacies in the symptoms assigned to aortitis, and its extreme obscurity as an abstract disease. It is unnecessary now to retrace the same ground, as M. Bouillaud himself has, in his later work, suppressed aortitis and its supposed symptoms, and judiciously concentrated his attention on its better half—endocarditis.

One form of acute arteritis may, however, be briefly glanced at, as an affection to which much attention has been directed by surgeons. I allude to what they have denominated, though with very questionable propriety, erysipelatous arteritis,—an affection which results from injury of an artery, as by a ligature, a gunshot wound, &c.—especially if there be deep-seated disease in the muscles of the affected part. The inflammation, in these cases, sometimes runs along the internal coat of the artery till it reaches the heart. It is stated to be a most formidable disease, rapidly producing great irritative fever, an extremely quick pulse, complete collapse, low delirium, and generally death. I suspect that the essence of this disease does not consist in its supposed erysipelatous nature, but in pus having found its way into the circulation, derived either from the deep-seated muscular injury, or from suppuration of the fibrinous coagulum in the injured portion of the artery. This is what we positively know to occur in phlebitis, the symptoms of which are identical with those assigned above to erysipelatous arteritis. That the local inflammation may, in the latter, be propagated to the heart, is consistent with analogy, because it is well ascertained that the same occurs in phlebitis. I have, in two or three instances, known venesection to cause endocarditis, and M. Bouillaud's five first cases of this disease, were connected with phlebitis (Traité, ii. p. 9). There is still another point in which the analogy is preserved: when a considerable artery is suddenly plugged up by coagula from inflammation, gangrene of the limb speedily ensues: if a great vein be similarly plugged, dropsy of the parts beyond is the result.

I have never seen or heard of a case in which inflammation, when confined to the interior of an artery, terminated in gangrene. Arteries, however, are frequently involved in the sloughing of surrounding parts; in which case, the blood generally coagulates in the vessels to a considerable extent above the line of sphacelation, and thus prevents hæmorrhage (Hodgson, p. 17).

Chronic Arteritis.—Arteries are more subject to chronic, than to acute inflammation. The internal membrane, when affected with it, is thickened, softened, and of a deep, dirty red colour. These appearances are not uniformly diffused, but are more marked in the vicinity of calcareous, steatomatous, and other degenerations. Hence some have supposed that these degenerations were the cause of the inflammation. There can be little doubt that they tend in many instances to keep it up; but it is highly probable that the degenerations themselves were originally caused by increased vascular action of a chronic nature. Since writing this in the first edition, I have seen nature engaged, as it were, in the very process. An intelligent student of St. Bartholomew's Hospital brought me a specimen of the aorta, in which the cellular tissue and fibrous coat were of a dim and pretty deep Indian red colour, with increased lacerability, behind patches and spots of the internal membrane, where opake, steatomatous yellowness, with thickening and elevation of the surface, were beginning to appear. There were also valvular vegetations. Acute rheumatism had preceded. Mr. Haydon guessed the age to be 22.

The appearances in arteries presented by chronic inflammation accompanied with morbid depositions, have been well known to authors from a very early period. The ancient physicians ascribed them to acrimonious, syphilitic, and scorbutic humours pervading the system. Some modern writers also, particularly Corvisart, Scarpa, Richerand and Hodgson, impute them to similar causes, especially to the syphilitic virus, or the mercury used for its eradication. After bringing the degenerations themselves under review, I shall revert to the consideration of their causes, as some difference of opinion on this point has existed amongst authors during the last twenty years.

Morbid alterations in the coats of Arteries, and especially the Aorta.—The morbid alterations in the interior of the aorta which appears to be of chronic formation, are, steatomatous, fibrous, cartilaginous, and calcareous depositions, with a thickened, fragile, and inelastic condition of the arterial coats: also ulcers and pustules.

Before describing the depositions, it may be premised that they originate, not in the internal coat, but either in the middle coat, or in the fine cellular tissue interposed between it and the internal coat; that the latter coat can sometimes be peeled off from them in a perfect state, even when they are far advanced; and that the productions themselves are more analogous to those of cellular and fibrous, than of serous membrane.

The extent, the form, and the thickness of the productions are infinitely various. Sometimes the several species exist separately, but, more commonly, they are found more or less intermingled in the same artery. The most simple morbid alteration is, a loss of elasticity, generally accompanied with increased density and opacity, of the coats of the artery. This state is sufficient of itself to give rise to dilatation, because (as will be more fully explained under the head of dilatation of the aorta) the elasticity and tone of an artery are the powers by which it resists the distending force of the blood.

The next, and the most common appearance, is that of small, opake, straw-coloured spots, immediately underneath the lining membrane, with slight inequality and corrugation of the membrane around them. At a more advanced period, the depositions form considerable, slightly elevated patches, which, becoming confluent, sometimes overspread the whole surface. Some of these patches have much the appearance and consistence of bee's-wax, or cheese, though in general their cohesion and flexibility are greater. These are usually denominated *steatomatous.* Others, presenting nearly the same colour, have a *fibrous* or ligamentous appearance; while others, again, are more translucent, white, and elastic, like *cartilage* or *fibro-cartilage.* I imagine that the steatomatous patches are merely imperfectly organised fibrine; that the fibrous are the same, more perfectly organised; and the cartilaginous, the ordinary transformation of the fibrous.

All the depositions described are accompanied with thickening and loss of elasticity of the internal coat, which becomes knotty, wrinkled, and sometimes cracked, scaly, and fimbriated. This state of the internal coat, however, is less marked before earthy depositions have taken place.

Earthy depositions generally commence in the midst of a cartilaginous or fibro-cartilaginous patch, though they are sometimes found in detached scales, and sometimes in the midst of steatomatous, cheesy, curdy, or melicerous matter, the softer varieties of which occasionally present the calcareous element, not in a concrete form, but plastic, like putty or mortar. When the earthy deposits form incrustations, the shape of these is irregularly flattened. Their external surface sometimes presents the imprint of the circular fibres of the middle tunic. Their internal surface is sometimes smooth, and evidently covered by the membrane; in other cases it is rough, and the membrane is more or less destroyed. Calcareous depositions are more frequent in the ascending portion and arch of the aorta, but, occasionally, they pervade the whole of the vessel, and even almost the whole of the arterial system. I saw a case in the Hôtel Dieu, in which the great arteries from the heart to the ankle were converted into rigid tubes by ossification, which, in parts, occupied all the coats and the whole circumference of the vessels. In another case, at St. George's Hospital, the common iliacs were rigid, and one, which was converted into a bony cylinder, was obliterated by a plug of dense lymph. The arterial system was elsewhere more or less ossified. Both the patients died with gangrenous sores of the legs,—the well-known result of ossified or otherwise obstructed arteries, and the consequent defect of local circulation.

In the arteries at the base of the brain, calcareous and other degenerations are remarkably frequent, and are a principal cause of rupture of the vessels, and apoplectic effusion. It is rare, indeed, to meet with instances of such effusion, exclusive of those from external violence, in which some disease of these arteries may not be detected. The arteries below the pelvic divarication of the aorta are more frequently ossified than those of the upper extremities and trunk.

Calcareous concretions differ essentially from natural bone. For, though some are formed by the secretion of the earthy phosphate in cartilage, even these have not the peculiar organised arrangement of bone. But, in by far the greater number of cases, the earthy matter is not secreted in any cartilaginous matrix, being simply deposited in the form of an irregular, homogeneous crust or crystallisation, without any determinate arrangement, and without vitality. The proportion of animal matter in these is very small. Mr. Brande found 100 parts to consist of 65,5 of phosphate of lime and 34,5 of animal matter. In some specimens I have found the quantity of animal matter considerably less.

When ossification is very considerable, it is sometimes attended with induration, inelasticity, and fragility, not only of the internal, but of all the arterial coats; and this state I have seen attended in some cases with thickening, and in others, though less frequently, with attenuation and a horny translucency of the walls of the vessel. In two cases of the latter, the walls were, in four or five small spots, as thin and transparent as a serous membrane. The aorta, so affected, generally undergoes dilatation, but very rarely contraction. When the depositions are partial and limited, the internal membrane in the intervals is often perfectly sound. This is especially the case in the ossifications of old people.

It is remarkable, that though morbid depositions are so frequent in the aorta, they are extremely rare in the pulmonary artery. Out of upwards of a thousand cases, in which I have examined this vessel, I have only once met with a calcareous deposition in its coats, (case of Lady R.,) and only three or four times with cartilaginous and steatomatous disease and dilatation.

Ulcers occurring in the arteries are, in general, a consequence of some previous chronic degeneration of the coats of the vessel. Such is the case when they are occasioned by the detachment of calcareous incrustations, or by the deposition of atheromatous or other matters underneath the internal membrane. Ulcers from these causes are not uncommon. They vary in size from that of a mustard-seed to that of a pea or bean, have more or less thick and ragged edges, and are sometimes so deep as to reach, and even to perforate, the external or cellular tunic. Laennec describes the formation of these ulcers from calcareous incrustations, in the following manner. "When a calcareous incrustation is detached from the aorta, the species of sinus left by it is filled up by fibrine, which becomes, by decomposition, of the consistence of friable paste, and is often intermixed with phosphate of lime." This paste, when soft and pulpy, has been denominated melicere or atheroma. Not unfrequently, the borders of the lesion are reddened for a little distance. Solutions of continuity, and ulcers connected with the detachment of calcareous incrustations, are among the most frequent causes of consecutive false aneurisms.

Small pustules, filled with pus, sometimes, though rarely, present themselves under the internal membrane of the aorta, and burst into its cavity. It is probable that they form the genuine or primitive ulcers of that vessel—those which are the most frequent cause of its perforation. They sometimes throw out curdy, and even calcareous matter. Laennec thinks that these pustules are occasioned by inflammation, not of the internal, but of the middle arterial tunic, or of the fine cellular tissue which unites the middle to the internal tunic. Pus is scarcely ever found on ulcers of the heart and arteries, because it is washed away as soon as secreted.

The Causes of Morbid Depositions in the Coats of Arteries.—Some authors, as M. Bouillaud, in his conjoint work with M. Bertin in 1824, have considered morbid depositions in the coats of arteries to be, in every case, the various metamorphoses of lymph, effused by inflammation. Others, again, of whom Laennec is the chief, have supposed that many, if not all, of the depositions in question, take place wholly independent of inflammation of any kind. As principles of treatment of a decided nature have been founded on each of these conflicting doctrines, it is a matter, not of mere speculation, but of practical importance, to examine the subject, and endeavour to ascertain the truth.

Analogical evidence derived from other membranes leads to the belief that chronic inflammation is, in most instances at least, the main agent concerned in the production of these depositions. Thus, for example, the dura mater, and the pleura or its subjacent cellular tissue, are sometimes not only thickened and indurated, but converted into fibrous, cartilaginous, or bony tissue. I have seen the dura mater converted into a calcareous plate nearly as large as the hand, and overspreading one hemisphere of the brain. The preparation was shown to me by my friend Professor Monro, and is in his museum. Mr. Hammick showed me two preparations in his museum of calcareous plates, of about two inches in diameter, on the pleura. Changes of this kind are, by general consent, attributed to chronic inflammation; as they are not only found in conjunction with organised adventitious membranes and other anatomical proofs of that form of inflammation, but are often attended with its symptoms. It is to be presumed, therefore, that corresponding changes taking place within an artery are referable to the same cause. That the morbid depositions in the artery should not be exactly identical with those found in other membranes, is to be anticipated on principles of general anatomy; for the effused matter, which is the basis of every accidental production, differs in aspect and nature according to the tissue in which it occurs. "Thus," as remarked by Bertin and Bouillaud, "serous membranes secrete a coagulable matter prone to transform itself into cellular or serous layers; the periosteum furnishes another matter, which concretes, hardens, and ossifies; the arterial tissue, composed essentially of a fibrous membrane, exhales a liquid which hardens, condenses, and becomes converted into cartilaginous patches, or calcareous scales."

But, admitting the agency of chronic inflammation as a cause of morbid alterations in arteries, there is reason to believe that some of them, particularly the calcareous, may take place independent of it.* For they are found in most old people; they sometimes occur in various detached points very remote from each other; they often consist of a simple calcareous scale, or an opake yellow spot, without any morbid state of the surrounding membrane; and such alterations almost always take place without affording the slightest sign, either general or local, of their formation. Now it is scarcely possible to conceive of an inflammation which manifests no symptoms, which is restricted to isolated points often remote from each other, which leaves none of the ordinary vestiges of inflammation in the surrounding parts, and which is the most frequent at that period of life when phlogistic action is the least prevalent. We are brought, then, to inquire what is the cause of morbid depositions when they do not appear to be referable to inflammation.

Here it is necessary to proceed with caution, as the ground is purely speculative. Laennec, indeed, thinks it the most simple and philosophical to acknowledge that we know not the nature of the derangement of the economy which produces an ossification or a cancer, but that very certainly it is not the same as that which produces pus—as inflammation (De l'Auscult. tom. ii. p. 684). If we are not satisfied to remain in this circumspect uncertainty, we can perhaps scarcely venture farther, in the actual state of our knowledge, than to suppose that morbid productions are sometimes results of a depraved action of the vessels, not identical with, or not amounting to inflammation—a doctrine, indeed, which rests on the basis of sound observation, and which has been extensively received since the accurate researches of the present century have bred a "philosophic doubt" on the tenet of the ancients, that all accidental productions are the effects of inflammation. Admitting a depraved action of the vessels, it is rational to suppose that, like inflammation, it would derive its particular character from the tissue which it affects: hence, that the fibrous and cellular tissues of the arteries might degenerate into cartilage, bone, &c.—the changes to which those tissues are prone under the influence of inflammation.*

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* The whole of this argument stands as in the original edition. The reader still sees that I have not, as M. Bouillaud avers, denied the intervention of inflammation.

But what is it that calls this depraved action into activity? It appears to me that over-distension of the arteries and their valves by the force of the circulation is what, principally at least, produces the effect. To this opinion I am led by the following considerations: 1. That it is very uncommon to see considerable hypertrophy with dilatation of the heart unattended by fibrous thickening of the mitral valve and its tendinous chords, though the valve be otherwise sound and efficient, and though no signs of inflammation had preceded: 2. That (according to an observation of Boerhaave, related by Morgagni) arterial ossifications are found in stags long and often exercised in running, and not in those which lead a tranquil life in the parks of the great: 3. That diseases of arteries and aneurism are more common, in the proportion of at least seven or eight to one, in men than in women, the life of the former being much more laborious, and the circulation more liable to excitement from potation of vinous or spirituous liquors, &c.: 4. That ossifications, &c. occur in those arteries, more especially, which are most exposed to over-distension; namely, the arch of the aorta, which immediately sustains the whole brunt of the left ventricular contraction, and the arteries of the brain, which, not having the support of a cellular sheath, and being bedded in a soft, pulpy substance, are weaker than any others: 5. That the arteries of the brain are more apt to become ossified when there is disease of the heart increasing the pressure upon them, either by increased afflux, as in hypertrophy, or diminished efflux, from venous retardation, as in dilatation or valvular obstruction: 6. That arterial ossifications are more especially incident to the aged, in whom the arterial and all other tissues sustain a diminution of elasticity and cohesiveness in consequence of the diminished vascularity which characterises old age. Perhaps the same reason, viz. over-distension, may be assigned for the remarkable frequency of the arterial depositions in those who have suffered much from syphilis or mercury; for as these maladies induce a cachectic state, which lessens the elasticity of all the tissues, the arterial tissue would, under these circumstances, suffer proportionably more from the distending pressure of the circulation. To the above catalogue we may perhaps adds gout, (an affection which is remarkably often attended with ossifications;) for, in this disease, there is not only a morbid condition of the general system, evinced by the deposition of gouty concretions in the fibrous structures; but there is also, in general, a morbid degree of plethora, and therefore a greater than ordinary prevalence of vascular tension.

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* I have the satisfaction of finding an identical train of reasoning in the Path. Anat. of Andral, though I had not seen his work when the above was written in 1831. After showing that, from the embryo to extreme old age, the fibrous and still more the cartilaginous tissues, present a constant tendency to ossification; that, in old age, ossification acquires a new disposition to seize on other parts of the fibrous and cartilaginous tissues; and that "*irritation or increased vascular action*" generally precedes osseous transformation when taking place at a premature period, or in parts where it does not usually occur in the progress of life, he proceeds:—"But in a variety of other cases, no morbid action whatever can be discovered previously to the deposition of osseous matter. How often, for example, do we find these depositions in the middle coat of arteries, in the fibrous tissue situated at the different orifices of the heart, in the dura mater, the pericardium, the capsule of the spleen, &c., without our ever having had the slightest evidence of the existence of any antecedent irritation of the part, either from the examination of symptoms during life, or of the morbid appearances found after death. No doubt, it may be argued that the irritation might have existed in a latent form; but before I can adopt such an hypothesis, *it must first be clearly and satisfactorily proved that this irritation is an essential element in the production of ossification;* in which case I must of necessity admit its existence, for then, the effect being produced, its cause must have existed either in a manifest or latent form. In my opinion, however, we have no more ground for admitting an increase of vitality as the cause of the osseous, than of the fibrous or cartilaginous transformation. We learn from observation that the nutrition of the part is perverted, and altered from its natural type, but neither theory nor observation shows any necessary connexion between this alteration and the exaltation of the vascular action of the part" (Vol. i. p. 370).

In the passage of the above quotation in italics, M. Andral, no less fairly than logically, throws the *onus probandi* on M. Bouillaud, and furnishes a complete reply to the following favourite argument of that author. "The first difficulty to be resolved was, to show by accurate cases that old men, affected with ossification, had not experienced any chronic, obscure, *latent* inflammation in the parts where the accidental productions were seated. But I declare that I have not found in any author the solution of this first and very grave difficulty. . . . . I think, in fine, that amongst the ossifications of the heart and arteries in old men, some have certainly been preceded by an inflammatory process, as in young subjects. But I declare that I do not yet possess all the necessary proofs to be convinced that there are others, in the production of which that process has positively not played any species of part Let us wait" (Traité, vol. ii. 308).

I might now corroborate the preceding argument respecting the occurrence of ossifications &c., independent of inflammation, by referring to the laws of analogous transformations, and showing that transformation of cellular tissue to fibrous, of fibrous to cartilaginous, and of cartilaginous to osseous, are frequent and common results of tension, friction, or increased exercise of the natural function of a part, wholly independent of inflammation. But I leave M. Bouillaud to do this for me, because, in the following *admissions*, where this distinguished author now qualifies his original opinions, which disclaimed any agency but inflammation, the reader will distinctly discern a recognition of my own argument. "It appears to me probable," says he, "that the perpetual friction to which the valves and arterial walls are subjected, is really a physiological or functional condition which ought not to be overlooked, in determining all the circumstances calculated to favour the development of certain indurations of these parts, whether cartilaginous, or osseous. No physician is ignorant that habitual pressure and friction on other parts, eventually entail various kinds of induration. Who knows not, amongst others, the horny excrescences (corns) produced on the toes by the pressure and friction of tight shoes? Who knows not the callosities of the hands in persons devoted to the most fatiguing manual occupations? Who knows not, finally, that tendous and fibrous tissues, subjected to long and violent friction, not unfrequently ossify? *Assuredly, it is not I who will deny the intervention of such causes* (Traité, ii. p. 309).

In admitting that these structural changes may occur independent of inflammation, M. Bouillaud admits as much as I have ever contended for in this volume, as much as Andral contends for; and as much as, in my opinion, completely cancels his own declaration at the conclusion of the preceding note.

The whole subject may be thus summed up. Organic diseases of the interior of the heart and arteries are, in general, results of inflammation; but it has not been proved that inflammation is, in all cases, an *essential* element in their production; and there are the strongest analogical reasons for believing that in some cases they occur independent of it.

Of the symptoms and treatment of chronic arteritis it is sufficient to say that, though it in all probability deteriorates the general health, it presents no distinctly appreciable signs but those of the structural alterations—the depositions, dilatations, and valvular obstructions, to which it gives rise. These signs are treated of under the heads of aneurism of the aorta and valvular disease.

PART III.

ORGANIC AFFECTIONS OF THE HEART AND GREAT VESSELS.

THIS part will comprise the organic diseases, first, of the muscular substance; secondly, of the pericardium; thirdly, of the internal membrane and valves; and fourthly, of the aorta. Adhesion of the pericardium has been noticed in Part II. for reasons there assigned. (See p. 191)

CHAPTER I.

HYPERTROPHY OF THE HEART.

SECTION I.

ANATOMICAL CHARACTERS, WITH CLASSIFICATION AND NOMENCLATURE OF HYPERTROPHY.

HYPERTROPHY is an augmentation of the muscular substance of the heart, resulting from increased nutrition.

As late as the year 1811, this affection was very imperfectly understood. No other form of it had been recognised, than that which was denominated by Corvisart *Active Aneurism*, (the *hypertrophy with dilatation* of Laennec,) a combination of two distinct affections which may exist independently of each other. Morgagni,* Corvisart,† and Burserius,‡ indeed, had each seen and described hypertrophy *without* dilatation; but it had not particularly arrested their attention, nor led to any inferences. It was reserved for M. Bertin in 1811 to throw new light on this subject. In three memoirs presented to the Académie Royale des Sciences, he proved that hypertrophy might exist, not only with dilatation, but also without it; that is, with a natural, and even with a diminished size of the cavity. Since that epoch, the concurrent observations of other pathologists, both abroad and in this country, have confirmed the accuracy of his observations, and led to the substitution of a new and more definite classification and nomenclature, in place of the inaccurate distinctions into *Active* and *Passive Aneurism* introduced by Corvisart.*

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* Epist. xvii. art, 21.

† 3d Edit., p. 335.

‡ Instit. Med.

Hypertrophy presents the following varieties.

1. *Simple Hypertrophy*, in which the walls are thickened, the cavity retaining its natural dimensions.

2. *Hypertrophy with Dilatation.* This, (the *eccentric* or *aneurismal hypertrophy* of Bertin,) presents two varieties: viz.

A.—With the walls thickened, and the cavity dilated.

B.—With the walls of natural thickness, and the cavity dilated: i. e. *hypertrophy by increased extent of the walls.*

3. *Hypertrophy with Contraction.* In this, (the *concentric hypertrophy* of Bertin,) the walls are thickened, and the cavity is diminished.

This classification is no less convenient than conformable to nature. The form B. of the second variety was not known to Laennec, though it was to Bertin. That it *really* consists of an augmentation of muscular substance, and therefore constitutes hypertrophy, is too manifest to require comment; but a further proof than mere structure is, that it sometimes produces the symptoms of hypertrophy,—a fact which the writer ascertained and made known in 1824, before he had any knowledge that M. Bertin had done the same.†

The terms "*eccentric* or *aneurismal*," and "*concentric*," are not so simple and expressive as *hypertrophy with dilatation* introduced by Laennec, and its natural converse *hypertrophy with contraction.* There is a further objection to the nomenclature of Bertin. His first variety of dilatation, though identical in its nature with his second variety of hypertrophy, is designated by a totally different name, viz. *active aneurism* (Bertin, p. 376); which could scarcely fail to lead the inexperienced student into the erroneous idea, that there was a difference in the nature of the two affections. Now the only difference consists in degree—in a predominance of the hypertrophy over the coexistent dilatation, or the converse. The terms, therefore, should be such as distinctly to imply identity in nature, and difference in degree only; and this is done in the simplest manner by giving precedence to the word *hypertrophy*, or *dilatation*, according as the one affection or the other predominates. Thus, *hypertrophy with dilatation* denotes a predominance of hypertrophy, while the converse *dilatation with hypertrophy* (vid. Dilatation) denotes a predominance of dilatation. *Hypertrophy by increased extent*, (without altered thickness,) *of the walls*, (the form B. of the second variety,) is thus designated when it is accompanied with the symptoms of hypertrophy, which I have observed to be generally the case when the patient is youthful and robust, and the disease has not made great advances; but it is called *simple dilatation* when the symptoms are those of dilatation, which is the case in aged or enfeebled subjects, or when the disease has made great advances.

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* Baillou and Lancisi were the first who applied the term *aneurism* to the heart: Morgagni and Corvisart followed, though they thought the application far from being correct. M. Bouillaud, notwithstanding, declares his adhesion to it (Traité, ii. 524). In my opinion, it is inadmissible, as it is to *dilatation*, and not to *aneurism*, (in the ordinary acceptation of the terms,) that an enlarged heart presents an analogy. But when a heart offers a *local or limited pouch or sac*, the analogy is to aneurism, and I reserve the term for these cases.

† Vid. an Essay by the writer in 1824, read to the Royal Med. Soc. Ed. The Treatise of MM. Bertin and Bouillaud was published in the same year.

I have thought it necessary to speak thus particularly on the subject of nomenclature, as, up to the present moment, it has created much confusion, and must continue to do so until the terms *active* and *passive aneurism* are forgotten.

Natural Dimensions and Weight of the Heart.—Before describing the anatomical characters of hypertrophy of the heart, it is necessary to give the reader an idea of the natural dimensions of this organ. Unfortunately, it is impossible to determine these positively; for, as they vary according to age, sex, and other circumstances, there is no immutable standard of comparison which might serve as a criterion. It is only by the eye, therefore, (and an experienced eye is necessary for the purpose,) assisted by approximative weights and measures, that it can be determined whether the proportion of the heart to the system, and of its several parts to each other, are natural. I shall first give the proportions according to Laennec, then subjoin the weights and measurements more recently made by M. Bouillaud, and finally add the still more recent results of Dr. Clendinning, my successor as physician to the St. Marylebone Infirmary.

The proportions assigned by Laennec approach perhaps as near the truth as it is possible to arrive. They are as follows: "The heart, comprising the auricles, ought to have a size equal to, a little less, or a very little larger than, the fist of the subject. The walls of the left ventricle ought to have a thickness a little more than double that of the walls of the right: they ought not to collapse when an incision is made into the cavity. The right ventricle, a little larger than the left, and having larger columnæ carneæ notwithstanding the inferior thickness of its walls, ought to collapse after an incision has been made into it. Reason indicates, and observation proves, that, in a sound and well-built subject, the four cavities of the heart are, within very little, equal to each other. But as the walls of the auricles are very thin, and those of the ventricles have much thickness, it results that the auricles form scarcely a third of the total volume of the organ, or the half of that of the ventricles." In the fœtus and very young children, the thickness of the left ventricle does not exceed that of the right to the extent described.

The right cavities are rather larger than the left, and this is not owing to sanguineous distension attendant on dissolution: for the disparity is found, though in a less degree, in animals destroyed by hæmorrhage.

The weights and measurements of M. Bouillaud have not, as this able observer frankly avows, been made on a sufficient number of subjects to warrant implicit confidence in the results; but they may be referred to with advantage while we wait for corrections from more extended observations. They are as follows:—

"In an adult of a medium height and well built, the mean weight of the heart is from 8 to 9 ounces; the mean circumference of the organ, at its base, is from 8 to 9 inches; the mean longitudinal and transverse diameters are 3½ inches; (the transverse diameter, in general, rather exceeds the longitudinal;) the mean antero-posterior diameter is about two inches.

The mean thickness of the walls of the left ventricle, at the base, is from 6 to 7 lines.

The mean thickness of the walls of the right ventricle, at the base, is 2½ lines.

The mean thickness of the walls of the left auricle is 1½ lines.

The mean thickness of the walls of the right auricle is 1 line.

The ventricular cavity, on an average, will contain a hen's egg, but the cavity of the right ventricle a little exceeds that of the left."

Dr. Clendinning favoured me, at my request, with the following summary of his researches :

" M. Bouilland's results seem to me to need rectification. The oldest subject of his first series, or that of health, appears to have been but 45 years; 8 of the 20 were 21 years and under; 3 were females. So that his healthy average can only apply to the period between 16 and 45, or, excluding females, 16 and 38. Now, if the heart increases with years, and, in the male, up to extreme age, and if disease of the heart be pre-eminently a disease of advanced years, it seems clear that Bouillaud's standard (8 to 9 ounces) will not serve for subjects mature or declining. I have attempted to obtain averages accommodated to the advancing development of the organ, of which a summary account is given in the Brit. Med. Almanac for 1838, p. 126.

Average weight of the heart.

	Males.	Females.
15 to 30	$8\frac{1}{4}$ oz.	$8\frac{1}{7}$ oz.
30 to 50	$8\frac{1}{2}$ oz.	$8\frac{1}{2}$ oz.
50 to 70	$9\frac{1}{3}$ oz.	8 oz.
70 and upwards	$9\frac{3}{4}$ oz.	8 oz.

Bouillaud has no measurements of bulk or specific weight:— I, none of linear dimensions, for reasons stated in my Croonian Lectures."

Anatomical Characters of Hypertrophy.—The muscular substance in hypertrophy is usually firmer and redder than natural. These characters, however, are not essential to the disease; for, in aged, or exhausted, anæmic subjects, the opposites are often observed; namely, flabbiness with paleness,—the states which prevail in the universal muscular system. When firmness exists in a great degree, it constitutes *Induration*, a distinct affection, dependent, not on increased, but on *altered* nutrition of the part, the elementary particles being denser than natural. It is generally attended with hypertrophy.

Hypertrophy may either be confined to a single cavity, or may simultaneously affect several, or even the whole. Sometimes one cavity is thickened, whilst another is attenuated. The full consideration of this subject comes under the head of exciting causes, as it is principally by these, that the nature and extent of the affection is determined. It may here suffice to remark, generally, that the ventricles are more obnoxious to the disease than the auricles, because they are exposed to a greater variety of exciting causes, and because the auricles are remarkably protected by the auriculo-ventricular valves.

When all the cavities are hypertrophous and at the same time dilated, the heart attains a volume, two, three, and occasionally even four times greater than natural; and its weight, properly 8 or 9 ounces, may be thrice as much (Bouillaud's weights). A case lately occurred at St. George's, in which it was two pounds and a half. The form of the organ, instead of being oblong, is then spherical, or even much broader than long, its apex is scarcely distinguishable, and, as the diaphragm does not retire sufficiently to yield space downwards for the enlarged organ, it assumes an unnaturally horizontal position, encroaching so far upon the left cavity of the chest, as sometimes to force the lung upwards as high as the level of the fourth rib, or even higher. I lately examined a subject in which it had been forced much higher. In Bouillaud's case 53, the base of the heart ascended to the second intercostal space, and its point reached the eighth! When great enlargement is accompanied by adhesion of the pericardium, the organ is secured by the attachments of the membrane, in a higher situation than its gravity would otherwise dispose it to assume; and being thus impacted between the spine and the anterior parietes of the chest, it is apt to occasion a preternatural prominence of the præcordial region. I am not aware that this remark has been made by any other writer, but I have seen the phenomenon in so many instances that I am disposed to assume it as a general fact. I have also shown that an increased, *double jogging impulse* results from the same cause (see Adhesion of the Pericardium, p. 194).

The *left ventricle*, being more prone to thickening, and not less to dilatation than the right, sometimes attains a volume seldom or never acquired by the right; and when its enlargement is enormous, it occupies not only the left præcordial region, but extends far under the sternum, where its impulse and sound may be mistaken for those of the right ventricle* (Case of Lambert).

The walls of the left ventricle, the natural thickness of which averages about half an inch in the adult, may be increased to the extent of one, one and a half, or, according to some, of two inches. M. Bouillaud's recent measurements place the range of hypertrophy of this ventricle between 7 and 14 lines. The cases are rare in which it exceeds an inch and a quarter, or 15 lines (see Figs. 15 and 20). The situation of the greatest thickening is usually a little above the middle of the ventricle, where the columnæ carneæ are inserted. Thence, the thickness decreases rather suddenly towards the aortic orifice, and gradually towards the apex, where it is reduced to less than half. When hypertrophy maintains these proportions in the different parts of the ventricle, the state is only an exaggeration of the natural form. The case is different when the hypertrophy takes place inwards and diminishes the cavity; for then the whole ventricle is nearly equally thickened, and its form is unusually globular.

The columnæ carneæ generally participate in hypertrophy, (Fig. 20,) but sometimes, when there is much dilatation also, they appear to be stretched, flattened, and attenuated. The interventricular septum, though belonging almost entirely to the left ventricle, is commonly less thickened than the external walls. When the left ventricle is greatly enlarged, the right, if unchanged, is applied, in a flattened form, to its superior and lateral part, and by contrast looks singularly small. But if, as generally happens, the right is elongated, it is, as it were, folded around the left.

The cavity of the hypertrophous left ventricle is sometimes dilated to such a degree as to admit the largest orange or the fist of an adult (e. g. Bouillaud's cases 132-62). I have twice, during the last six months, seen it exceed even these dimensions. On the contrary, in hypertrophy with contraction, the cavity may be reduced to the size of a small walnut or a pigeon's egg: in Bouillaud's case 118, "it could scarcely contain the finger." These are the extremes of dilatation and contraction. As the natural capacity of the left ventricle averages the size of a hen's egg, it may be regarded as considerably dilated when it equals that of a goose's egg.

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* Laennec, tom. ii. p. 507.

When the *right ventricle* alone is hypertrophous, it may descend lower than the left, and constitute the apex of the heart. Its columnæ carneæ, naturally more numerous and complicated than those of the left, are more susceptible of thickening than the walls themselves of the cavity. Hence, the increased size of the columnæ is commonly the first object that arrests the attention, and to them alone is the hypertrophy in many instances confined. They are sometimes so curiously interlaced and attached, as to traverse the ventricle in every direction, subdivide it into various compartments, and in some cases, almost totally to fill up its cavity, as in case 89 by Bertin, and that of Collins. These changes never take place to the same extent in the left ventricle. The total thickness of the walls of the right ventricle, naturally averaging 2½ lines, rarely exceeds four or five; yet it has been known to attain from eleven to sixteen, as appears from the 88th case of Bertin, and one, by Soins, in the *Archives de Médicine.* In Bouillaud's case 76, it was 8 to 10 lines, and in case 77 about an inch. In a girl of nine years old, (see case of Collins,—Cyanosis,) I have met with it measuring six or seven lines; which is equal in proportion to nearly double that extent in the adult. The greatest thickening of the right ventricle is near its base: lower down, though the columnæ carneæ be enlarged, their interstices are usually thin, and not unfrequently translucent.

The cavity of the right ventricle, naturally a little larger than a hen's egg, may be dilated to the size of a goose's egg or more; or it may be contracted to less than a pigeon's egg. In Bouillaud's case 65, it would scarcely contain the thumb, and in case 123, the columnæ carneæ were so thickened and adherent that there was scarcely any cavity left, and the blood could only filter through the narrow spaces between them. These small dimensions are generally in connexion with malformations of the heart, and, especially, the open foramen ovale and contracted pulmonic orifice.

Hypertrophy may not only be confined to a single ventricle, whether the right or the left, but it may be confined to particular parts only, as the base, the septum, the apex, the columnæ carneæ, or the external walls; the remainder of the cavity being either natural, or attenuated. Again, a thickened ventricle may be contracted in one part, while it is dilated in another. In examining in the dead subject mixed cases of these descriptions, it is necessary to counterpoise the opposite conditions, to balance the hypertrophy against the tenuation, and the dilatation against the contraction, in order to determine which is the predominant affection.

The *hypertrophy of the auricles* is almost invariably of the second species, or that with dilatation. Laennec even states that he has never met with any other (Laennec de l'Auscult. tom. ii. p. 524). The *simple* and the *contracted* forms, however, are not without example. The thickening is diffused in a very uniform manner throughout the cavities, the musculi pectinati being the only parts in which it is more considerable than elsewhere; and, as they are larger and more numerous in the right, than in the left auricle, it is in the former that hypertrophy proceeds to the greatest extent. It occasionally renders the auricle nearly as thick as the right ventricle. This I have never known to take place in the left auricle. Sometimes the musculi pectinati are the only parts in which hypertrophy shows itself. The thickening of the auricular walls seldom exceeds double the natural state, (*i. e.* 1½ lines for the left auricle, and 1 for the right,) and, being even then inconsiderable, it may easily be overlooked by an inexperienced eye. When it amounts to a quarter of an inch, which is rarely the case, it is very perceptible.

When hypertrophy has been preceded by pericarditis or endocarditis, it is common to find the ordinary vestiges of inflammation: namely, adhesion or other changes of the pericardium, and thickening, with opacity, of the valves and tendinous chords, from hypertrophy of their fibrous tissue, and its transformation into steatoma, cartilage, or bone. These valvular changes may also take place independent of inflammation, as shown at p. 227. So common, indeed, is the fibrous transformation, that in cases of great hypertrophy with dilatation, though the valves retain their natural size and efficiency, they are very rarely exempt from thickening; as if they required to be strengthened in order to sustain the augmented force of the ventricle, and as if the increase of action resulting from this force, was, in conformity with a general law, the cause of their hyper-nutrition.

SECTION II.

MODE OF FORMATION WITH THE PREDISPOSING AND EXCITING CAUSES OF HYPERTROPHY.

Mode of formation and predisposing causes of Hypertrophy.— I shall first notice this affection as resulting from ordinary causes, and finally advert to its connexion with inflammation.

Hypertrophy, independent of inflammation, takes place in the heart by the same process as in any other muscle. Increased action causes an augmented afflux of blood, and there results a corresponding increase of nutrition. Diminished action, on the contrary, has the reverse effect. Thus, the arms of the smith and the legs of the dancer, are unusually robust; while limbs paralysed or not exercised, are pale and emaciated. If, however, the circulation can be reinvigorated in the palsied part, nutrition is increased. An individual within my knowledge, whose arm had, in consequence of an attack of hemiplegia, been for twenty years emaciated, contracted, without radial pulse, and immovably fixed to the side, submitted the limb to the process of vigorous shampooing. In a few months, the pulse returned, the emaciation sensibly diminished, and the motive power was so far restored that the individual could raise the hand above the head.

In the same way, when, from mechanical obstruction or any other cause, blood is inordinately accumulated in the heart, the organ is provoked to extraordinary efforts; it struggles against the obstacle; it frets and labours to overcome it; the coronary arteries are excited to increased activity: augmented nutrition ensues; the parietes are thickened, the muscular power is increased; the effects, superadded to the cause, induce a still greater violence of action; and, thus, the disease is not only established, but has a constant tendency to increase.

The left ventricle is much more prone to hypertrophy than the right; and the right, again, than the auricles.

This admits of explanation on very simple principles. It is found that hollow muscles resist over-distention by their contents with a force exactly proportionate to their strength. Now, as the act of resistance, by stimulating the arteries to increased action, is the cause of increased nutrition, it follows that stronger muscles must be the more susceptible of hypertrophy. Accordingly, on referring to the heart, we find that the relative structure of its several compartments is such as to predispose the organ to those changes which it actually undergoes from over-distention.

The left ventricle, for example, being charged with the immense burden of the greater circulation, is proportionably substantial and robust; the right, having the comparatively light task of propelling the blood through the minor or pulmonary system, is little more than one third as thick and powerful as the left: the auricles, again, having a still less laborious function to perform, have a still more limited muscular provision.

Hence, it is easily understood how a distending force sufficient to overcome the contractile and elastic power of the right ventricle, might merely operate as a stimulus to the superior muscularity of the left. While the former, therefore, incapable of reacting on its contents, would dilate; the latter, excited to extraordinary efforts, would become hypertrophous.

It is not, however, to be supposed, that while the left ventricle is becoming hypertrophous, it may not, at the same time, undergo dilatation: nor, on the other hand, that the right ventricle, while yielding to dilatation, may not become hypertrophous; for observation teaches us, that the combination of hypertrophy with dilatation, either in the left ventricle alone, or in the two conjointly, is the most ordinary form of organic disease of the heart.

For an explanation of the cause why dilatation accompanies hypertrophy, the reader may refer to the chapter on dilatation. Why hypertrophy sometimes accompanies dilatation of the right ventricle, may be here explained, and it admits of an explanation in one or other of two ways.

1st. It has been remarked by Laennec, (Traité de l'Auscult. tom. ii. p. 496,) that a large proportion of mankind are born with ill-proportioned hearts, the parietes being a little too thin, or a little too thick, on one or both sides. Now, when this unnatural thickness exists in the right ventricle, it is clear from what has been said above, that it must impart to that ventricle an increased disposition to hypertrophy. This explanation, however, is not very satisfactory, as the existence of the malformation described by Laennec cannot be positively proved: yet, as all the other organs and parts of the body are liable to defects of natural conformation,—as, in other terms, the all-wise Author of nature, who operates by natural means, has sown the seeds of mortality in every part of the system, it is consistent with analogy to suppose that the heart may be liable to the same.

2dly. As augmented nutrition is excited in the left ventricle by stimulating it in proportion to its power, so a stimulus bearing the same proportion to the power of the right ventricle, must have the same effect on it also. Accordingly, in the majority of cases of hypertrophy of the right ventricle, an obstacle is found to exist of such a nature as the one described. The obstacles which I have most frequently found to produce the effect, are, contraction of the mitral valve operating in a retrograde direction through the lungs, and that of the orifice or semi-lunar valves of the pulmonary artery. These affections being usually slight at their commencement and slow in their progress, oppose an obstacle to the circulation not only moderate in degree, but constant in its operation,—the two circumstances best calculated to induce hypertrophy of the right ventricle. M. M. Bertin and Bouillaud conceive that the greater tendency of the left ventricle than of the right to hypertrophy, depends upon the more stimulant quality of the arterial blood circulating through the former. This opinion they found on the circumstance that hypertrophy of the right ventricle in most cases accompanies patescence of the foramen ovale, which lesion, they think, causes an influx of arterial blood into the right ventricle. But, admitting that arterial blood in the right ventricle does occasion hypertrophy, it does not follow that it should have the same effect on the left; for, of the former ventricle it is a morbid stimulus, but of the latter it is the natural one. Accordingly, direct proof is to be found in the auricles that arterial blood is not the cause of hypertrophy; for the left auricle, which, on M. M. Bertin and Bouillaud's principle, ought to be more subject to hypertrophy than the right, is less so. It will be shown, moreover, in the chapter on malformations of the heart, that, in the cases on which these gentlemen found their opinion, the blood does not enter the right ventricle.*

Exciting causes of Hypertrophy.—According to the foregoing opinions on the mode of formation of hypertrophy, it will be apparent that every circumstance capable of increasing the action of the heart for a sufficient length of time, may be a cause of hypertrophy. These circumstances may be either of a nervous, or of a mechanical nature.

1. The nervous class comprises all moral affections and all derangements of the nervous function that excite long-continued palpitation.

2. The mechanical class embraces all physical causes which can either accelerate, or obstruct the circulation, and thus occasion a preternatural pressure of the blood upon the heart.

The physical causes which accelerate the circulation, are, violent and protracted corporeal efforts of every description. In growing youths, excessive rowing is one of the most efficient. I have met with numerous instances in which it has produced the effect,—especially in Oxford and Cambridge men, forming the crews of the racing boats. In schoolboys, I have found violent gymnastics, the game of "hare and hounds," and actually following the hounds, produce the same effect. These violent exercises may even occasion rupture and inflammation of the valves and aorta, issuing in incurable organic disease,—of which I have seen several well-marked instances. I have also repeatedly known pedestrian tours amongst the Swiss and Scotch mountains, to be followed by hypertrophy and other diseases of the heart. It is *protracted* efforts that are always the most pernicious. Feats of this kind should, therefore, always be discouraged.

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* M. Bouillaud, who drew up the work of Bertin, was the author of the above opinion. In his Treatise in 1835, (vol. ii. p. 456,) he abandons the idea that the stimulant quality of the arterial blood predisposed the left ventricle to hypertrophy, and he "avows that this cause of *irritation* or of *excitation* is a little hypothetical" in reference even to the right; yet he thinks that the hypertrophy of the right, which usually accompanies a communication between the two ventricles, and the hypertrophy of the coats of veins in cases of *varicose aneurism*, are strong considerations militating in favour of his opinion that the irritation of arterial blood may contribute to occasion the hypertrophy. It must not, however, be forgotten that, in both the preceding cases, the *weight* of the arterial circulation is thrown on the venous system, and it may be asked whether this alone is not sufficient to account for the hypertrophy, both of the right ventricle and the veins.

The physical causes which obstruct the circulation are very numerous. They comprise smallness of the aorta, whether congenital or acquired; dilatation of the aorta; inequalities of its internal surface; all diseases of the valves of the heart which either contract their apertures, impede their movements, or allow of regurgitation; adhesion of the pericardium; all affections of the chest that for a long period obstruct the circulation through the lungs, as chronic catarrh, emphysema, asthma,* narrowness of the chest, either congenital, or occasioned by curvature† of the spine, &c.; encroachment of the diaphragm on the cavity of the chest from the pressure of the gravid uterus, of ovarian dropsy, of other abdominal tumours, but, perhaps above all, of long, stiff stay-bones or wooden *busks*, which, by fixing the abdomen, prevent the descent of the diaphragm, and, when the abdomen is flatulent, act with the power of a long lever in depressing the sternum. The effect takes place *even though the stays be not very tightly laced*, whereas a pretty tight band round the waist will be borne with impunity, provided that the chest and abdomen can expand freely above and below it.

Such are the ordinary predisposing and exciting causes of hypertrophy. There is strong reason to believe that inflammation is another cause. For the last eight or ten years, I have almost invariably found, that palpitation following acute rheumatism was connected, either with a persistence of the inflammation in a chronic form, or with valvular disease or adhesion of the pericardium, resulting from it. As these latter lesions are, of themselves, capable of exciting hypertrophy, we cannot argue, from cases in which they exist, that the hypertrophy was referable to inflammation: we must first select, and argue from, cases in which the inflammation was *not* attended with organic lesions. Now, I believe that I have seen a certain number of cases of hypertrophy, which, though unattended by any valvular defect or adhesion of the pericardium, were distinctly dated from attacks of acute rheumatism, attended with inflammation of the heart. Hence, it is to be inferred, that in such cases, the inflammation *alone* was the cause of the hypertrophy. Assuming this as true, we may next argue back to those cases in which the inflammation has produced valvular and other organic lesions, and we may legitimately say that the inflammation, no less than the organic lesion, has contributed to the production of the hypertrophy. Accordingly, it is extremely rare to find hypertrophy absent in cases of valvular disease resulting from rheumatic endocarditis, whereas, it is not unfrequently absent in cases of valvular disease resulting from ordinary causes,—a result which might be expected, since, in the inflammatory cases, there is the co-operation of two causes,—the inflammation and the valvular lesion.

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* I have not found phthisis so decided a cause of disease of the heart as we should be led to suppose from the extreme pulmonary obstruction to which it sometimes gives rise. The reason of this appears to me to be, that, in the early stages, when the disorganisation is not extensive, the circulation is little embarrassed; and in the advanced stages, the mass of circulating fluids is so much diminished, in consequence of deficient nutrition and augmented cutaneous transpiration, that the heart sustains little additional burden from the obstruction in the lungs.

† The majority of hump-backed persons are ultimately attacked by disease of the heart.

These results of observation are countenanced by analogy; for there is no fact in modern pathology better established, than that chronic inflammation, (either originally chronic or consequent on acute,) may excite hypertrophy of various organs and tissues. Thus, Andral, after referring one class of hypertrophies "simply to increased exercise of the functions of the affected organ," refers another to "an acute, but more frequently to a chronic attack of hyperæmia (inflammation). In such cases, the hypertrophy is sometimes confined to the tissue which was originally in a state of irritation and hyperæmia; while, sometimes, after the tissue originally affected has returned to its natural, healthy condition, *the adjacent tissues* retain a chronic form of disease, and fall into a state of hypertrophy. Such is frequently the termination of inflammation of the skin and mucous membranes" (Path. Anat. i. 224). In the writer's Elements and Illustrations of Morbid Anatomy, are coloured delineations of hypertrophy, not only of the other tissues, but of the muscular coats of the stomach, colon and bladder, connected with chronic inflammation of the mucous membrane. I have seen the same in the bronchial muscles. It is very intelligible, then, that inflammation may be similarly propagated from the membranes to the muscular substance of the heart. The changes of colour and consistence, which the organ undergoes under these circumstances, are described in the chapter on *Softening.**

SECTION III.

ORDER OF SUCCESSION IN WHICH THE SEVERAL COMPARTMENTS OF THE HEART ARE RENDERED HYPERTROPHOUS BY AN OBSTACLE BEFORE THEM IN THE COURSE OF THE CIRCULATION.

As an obstacle to the circulation operates on the heart in a retrograde direction, the cavity situated immediately behind it is the first to suffer from its influence. Accordingly, all the impediments seated in the aorta, its mouth, or the arterial system, act primarily on the left ventricle, which being likewise exposed to the heaviest burden when the circulation is accelerated, has to conflict against a greater variety of exciting causes of hypertrophy, than any other cavity of the heart. On this account, therefore, as well as from the thickness of its parietes, it is subject to hypertrophy in a greater degree than any other.

So long as the left ventricle is capable of propelling its contents, the corresponding auricle, being protected by its valve, remains secure. Hence, in a large proportion of cases, the auricle is perfectly exempt from disease, while the ventricle is even enormously thickened and dilated. But when the distending pressure of the blood preponderates over the power of the ventricle, its contents, from not being duly expelled, constitute an obstacle to the transmission of the auricular blood. Hence the auricle becomes over-distended, and the obstruction may be propagated backwards through the lungs to the right side of the heart, and there occasion the same series of phenomena. When the obstruction thus becomes universal, as is frequently the case, it may either happen that all the cavities are thickened, or those only which, from their conformation, have the greatest predisposition to it.

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* Dr. Elliotson broached the opinion in 1830 that "hypertrophy was *in general* an inflammatory disease;" and his "reason for supposing so, was, that it was a very common effect of pericarditis" (Lum. Lectures, p. 25). I think that others in this country had previously entertained a similar opinion. I presume that it was not entertained in France, because M. Bouillaud, so late as 1835, introduces it as a novelty, (aperçus nouveaux,) of his own discovery (Traité, ii. p. 457). Dr. Elliotson has greatly overstated the case in saying that hypertrophy is *in general* an inflammatory disease: nor can I at all subscribe to his opinion that the inflammatory is the only curable variety of this disease (Med. Gaz., June 22, 1833, p. 377). On the contrary, I have found it the least curable, on account of the frequency and severity of valvular and other complications; whereas, uncomplicated hypertrophy, from ordinary causes, is, in a large proportion of cases, very curable by the treatment presently to be described.

When the mitral orifice is contracted, especially if the aperture be very small, the left ventricle, being insufficiently supplied with blood, is not stimulated to its ordinary contractile action, and consequently becomes emaciated and occasionally flaccid or softened. Meanwhile, the left auricle, having to struggle against the contracted valve in front, and also to sustain the distending pressure of the blood flowing in from the lungs, invariably becomes thickened and dilated. The engorgement, extending backwards through the lungs to the right ventricle, often occasions its hypertrophy and dilatation; under which circumstances, namely, hypertrophy of the right ventricle and contraction of the mitral valve, the lungs suffer in a pre-eminent degree: for, being exposed to the augmented impulsive power of the right ventricle behind, and incapable of unloading themselves on account of the straitened orifice in front, their delicate and ill-supported vessels are strained beyond the power of resistance. If, therefore, they cannot disgorge themselves sufficiently by a copious secretion of watery mucus, they effuse blood by transudation into the air-vesicles and tubes, and form the disease denominated *pulmonary apoplexy*. I have found this affection to occur more frequently under the circumstances described, namely, great contraction of the mitral valve, with, or even without, hypertrophy and dilatation of the right ventricle, than under any other.*

When the mitral orifice is permanently patescent, so that, at each ventricular contraction, blood regurgitates into the auricle, this cavity suffers in a remarkable degree: for it is not only gorged with the blood which it cannot transmit, but, in addition, sustains the pressure of the ventricular contraction. Permanent patescence of the mitral orifice, therefore, constitutes an obstruction on the left side of the heart; and the effect of this, as of contraction of the orifice, may be propagated backwards to the right side. The regurgitation is always considerable when it renders the pulse small and weak.

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* This fact has subsequently been corroborated by Dr. Wilson, in a paper, with cases, read to the College of Physicians. I have more recently found that softening of the heart is also a frequent cause of pulmonary apoplexy (See *Softening*).

When the impediment to the circulation is primitively seated in the lungs, the right ventricle, situated immediately behind them, is the first to experience its influence; and when the cavity is so far overpowered by the distending pressure of the blood as to be incapable of adequately expelling its contents, the obstruction extends to the auricle,—the process being exactly the same as that which I have already described, (p. 249,) in reference to the left ventricle and auricle.

Obstruction in the right auricle, whether from this or any other cause, presents an obstacle to the return of the venous blood, and therefore causes retardation throughout the whole venous system. Nor is this all; for the retardation is propagated through the capillaries to the arterial system, and thus at length returns in a circle to the heart. In this way is explained what at first sight appears an anomaly: namely, that the left cavities are sometimes rendered hypertrophous by an obstruction in the heart situated behind them in the course of the circulation, as, for instance, when the left ventricle is rendered hypertrophous by a contraction of the mitral orifice.

The reader must here be again reminded that the exciting causes of hypertrophy are equally those of dilatation; and that, supposing no unknown agencies to interfere, as may sometimes possibly happen, it depends on the proportion which the cause bears to the reacting energy of the cavity exposed to its influence, whether that cavity become affected with hypertrophy, with dilatation, or with a combination of the two.

It may be said, generally, that when congestion is *constant* in a cavity, dilatation is more commonly the result; and that when there is only resistance to the expulsion of the blood, without constant engorgement of the cavity, it is more common for hypertrophy to be produced. Contraction, for instance, of the aortic orifice, causes hypertrophy of the left ventricle in a greater degree than dilatation; whereas, patescence of that orifice, attended with regurgitation and constant engorgement of the cavity, causes dilatation in a greater degree than hypertrophy.

Hypertrophy with contraction most commonly proceeds from straitening of an orifice. Thus the greatest hypertrophy with contraction of the right ventricle upon record, was accompanied with straitening of the pulmonary orifice to two lines and a half in diameter (Case 87 by M. Bertin). I have met with a very similar case, and several, connected with malformation of the heart, are on record.

It may be useful to subjoin a list of the various forms and combinations of hypertrophy and dilatation, and to show the comparative frequency of their occurrence. On the latter point I shall offer the results of my own observation, and I believe that they correspond very closely with those of others.

The diseases are of more frequent occurrence in proportion as they are higher in the following scale.

1. Hypertrophy with dilatation of the left ventricle, and a less degree of the same in the right.
2. Hypertrophy with dilatation of the left ventricle, with simple dilatation of the right.
3. Simple dilatation of both ventricles.
4. Simple hypertrophy of the left.*
5. Dilatation with attenuation of the left.
6. Hypertrophy with contraction of the left.
7. Hypertrophy with contraction of the right.†

Of the Auricles.

1. Distention, particularly of the right, from congestion during the period of dissolution.
2. Dilatation with hypertrophy.
3. Simple hypertrophy.
4. Hypertrophy with contraction, which is almost unknown.

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* M. Bouillaud thinks that there is scarcely one case of *simple* hypertrophy of the heart in general, for twenty of hypertrophy with dilatation.

† M. Bouillaud gives eight cases of this for five of the same in the left ventricle, but he is not sure that further observation would establish the majority in favour of the right.

SECTION IV.

PATHOLOGICAL EFFECTS OF HYPERTROPHY, AND MODE OF THEIR PRODUCTION.

M. Laennec supposes the general symptoms of all organic diseases of the heart to be nearly the same (De l'Auscult. tom. ii. p. 487). It may be said, without prejudice to one who has done so much, that, on this subject, both he and all the authors who preceded him, have entertained inaccurate ideas. They had studied these diseases in the aspect under which they most commonly present themselves; namely, complicated one with another; and it is unquestionable that, when so viewed, they display a general similarity in their symptoms. But it had never occurred to those authors to analyse each disease in an isolated form. When so examined, although certain symptoms are common to all, they severally manifest differences of a striking kind, obviously dependent on their respective organic peculiarities, and which may, therefore, be fairly regarded as the essential and diagnostic characters of each.

M. Bertin has the merit of having been the first to display in a clear light the essential pathology of hypertrophy. His distinguished talent for generalisation, however, has, I believe it will be allowed, carried him a degree too far. He contends that authors are wrong in having assigned to hypertrophy or *active aneurism*, as its symptoms, dyspnœa, suffocation, violet injection of the face, engorgement of the lips and of the venous capillaries in general, passive hæmorrhages, and serous infiltration. He contends that these are the signs, not of hypertrophy, but of a coexistent lesion: viz. a contracted orifice, or any other affection capable of obstructing the circulation; and that pure, uncomplicated hypertrophy is characterised by signs of increased activity and energy of the circulation, instead of by dropsy and the other signs of its retardation.*

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* I now find that M. Bouillaud is the author of these opinions, as they are transplanted into his own more recent work, vol. ii. p. 445. Seven years of additional observation have only the more convinced me that they are erroneous.

That this is true in reference to the *pure, uncomplicated* form of the disease, *before embarrassment of the capillary circulation has taken place*, will not be denied by any one who has had opportunities of verifying the symptoms by dissection. But M. Bertin is not, in my opinion, supported either by sound observation or by analogy when he says that serous infiltration and the whole class of symptoms bespeaking an obstructed circulation, are totally foreign and repugnant to hypertrophy. The truth I believe to be, that the very same energy of the circulation which gives rise, as he admits, to active hæmorrhages, apoplexy, &c., causes, as its next effect, and in the more advanced stages of the disease, engorgement of the arterial capillary system; the necessary consequence of which is, serous infiltration and more or less of all the other symptoms indicative of retardation of the blood. The process appears, in fact, to be strictly analogous to that by which serous infiltration is produced in cases of erysipelas, inflammatory anasarca, acute rheumatism, &c. I would not be understood by this to mean that active capillary congestion is identical with inflammation, but that, as the effects of the two are sometimes the same, we are compelled to admit a close analogy in the mode of their production. It is now, indeed, very generally allowed that active congestion only differs from inflammation in being a degree less.

M. Bertin himself unconsciously shows that hypertrophy may produce an obstacle to the circulation, for he says that, when the heart is enormously enlarged, the respiration is impeded in a very eminent degree (d'une manière très notable. Bertin, p. 359). Now, what is the real cause of this impeded state of the respiration? He ascribes it to the encroachment of the heart upon the lungs; but this cause is inadequate: for tumours of a much larger size, as, for instance, aneurisms of the aorta, malignant tumours, &c. have existed in the chest, even for years, without producing similar inconvenience. It is not, therefore, to compression of the lungs that we are to look, as the cause of the dyspnœa and dropsy; but, clearly, to the heart itself;* and on reflection it is very conceivable that, when the blood is poured in increased quantity and with unwonted impetuosity into the capillary vessels of the lungs, so as to gorge and obstruct them, the obstruction *being universal*, must be greater than when a free channel is left open through a portion of the organ, even though limited, as one half or a third; for we constantly see, in cases of phthisis, and of chronic pleuritic effusion, that such a portion is sufficient for maintaining the circulation.

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* M. Bouillaud now admits the co-operation of this (Traité, ii. p. 445).

The primary effect of universal obstruction of the lungs by engorgement, is, to produce œdema of their cellular tissue, and dyspnœa. The secondary effect is, to gorge the right side of the heart, and thus impede the return of the venous blood from the system at large; which co-operates with the increased energy of the arterial circulation in producing capillary congestion and its consequence, anasarca.

It must be admitted, however, that hypertrophy does not produce serous infiltration so readily and promptly as a direct, primary obstacle to the return of the venous blood; a fact which admits of a rational and obvious explanation. When there is an obstacle to the return of the venous blood, suppose, for instance, contraction of the tricuspid, pulmonic, or mitral orifice, two causes conspire to produce the capillary congestion; namely, the direct pressure of the arterial vis-a-tergo, and the retrograde pressure of the retarded venous blood. But when the latter pressure does not exist, when the veins freely receive and transmit their natural proportion of blood, the force of the arterial circulation must be very greatly increased, before it can so far overcome the elasticity of the capillaries as to give rise to engorgement and infiltration.

This satisfactorily accounts for the difference in the history and character of infiltration as resulting, on the one hand, from pure hypertrophy, and, on the other, from contraction of a valve or other primary obstacles to the circulation, amongst which (for reasons to be explained in the chapter on dilatation) I include dilatation with attenuation, and softening. In the former case, it appears late, is generally moderate in extent, and requires for its production an aggravated form of hypertrophy; in the latter cases, it appears comparatively early, is more copious, and yields with less facility to remedies. It will, however, be shown in the chapter on valvular disease, that the dropsy seldom comes on in any considerable degree till hypertrophy, dilatation, or softening has been superadded to the valvular lesion.

The same reasons that account for the tardy occurrence of dropsy in pure hypertrophy, account, likewise, for another characteristic of this malady, when moderate in degree; namely, the slight and transitory nature of the attacks of dyspnœa. For, if the quantity of blood impelled into the lungs by the right ventricle, and the force with which it is impelled, are not very excessive, the pulmonary veins are capable of relieving the engorgement almost as quickly as it takes place, and, consequently, the hurry of the respiration subsides promptly after the removal of its exciting cause.

The sum, then, of all that has been said, is, that pure hypertrophy at first gives rise to increased force and activity of the circulation; and that, when this force surmounts the natural tonic power of capillaries, (which is apt to be the case in the late stages of the disease,) congestion, infiltration, and the other phenomena of an obstructed circulation, ensue.

To these principles an exception presents itself in hypertrophy with contraction, when the cavity of the ventricle is so small as to be incapable of transmitting the natural quantity of blood. In this case, supposing the left ventricle to be the one affected, the arterial circulation sustains a diminution of force and activity; and, whether the one ventricle or the other be affected, it creates an obstruction tantamount to that produced by valvular contraction, and, on the same principles, generates dropsy and the other phenomena of a retarded circulation. I have met with three or four cases in which the ventricle was reduced to the size of a small walnut.* Such cases, however, are very rare.

The effects of simple hypertrophy and hypertrophy with dilatation of the left ventricle, on the brain, are so pre-eminently important, that it is necessary to advert particularly to this subject, for the purpose of bringing it prominently into view.†

Since the researches of the present day have demonstrated that even a slight thickening of the walls of the heart constitutes a morbid state; and have unfolded to view the connexion subsisting between that state and a train of symptoms formerly either wholly overlooked or attributed to other causes; instances of apoplexy supervening upon hypertrophy have been so frequently noticed, that the relation of the two as cause and effect, is, in my opinion, one of the best established doctrines of modern pathology. Eight or nine cases of suddenly fatal apoplexy, and numerous cases of palsy, from hypertrophy, have, within a few years, fallen under my own observation. In the majority of them the patient exhibited what is commonly called the "apoplectic constitution;" that is, a robust conformation, a plethoric habit, and a florid complexion: in others, these characters were absent; but the total number of the cases of apoplexy from hypertrophy, is much greater than I have witnessed, during the same period, of apoplexy from causes independent of hypertrophy. Whence I am led to believe, with M. Richerand and M.M. Bertin and Bouillaud, that hypertrophy forms a stronger predisposition to apoplexy than the apoplectic constitution itself; and that, in most instances, those persons who present the apoplectic constitution in conjunction with symptoms of increased determination to the head, are, at the same time, affected with hypertrophy.

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* See one by the writer; Lond. Med. Gaz. Sept. 5, 1839. p. 422.

† I exclude hypertrophy with contraction, for the reasons mentioned in the preceding paragraph.

During the last eight years, I have had much additional reason for adhering to the same opinion. It has also been advocated by M. Brichteau, in the Clinique de l'Hôpital Necker, M. Bouillaud, and numerous other writers. It appears to me, indeed, that the *full* extent of the connexion between the diseases of the head and those of the heart,—in reference, not only to hypertrophy, *but also to dilatation, softening, and diseases of the valves,*—has not yet been duly estimated, either by the writers referred to, or by the general body of the profession.

From the 12th December, 1832, to the same date in 1834, 39 patients who had died of apoplexy, were examined *post mortem* in the St. Marylebone Infirmary, to which I was then physician. The following are the results, according to the journals of Mr. Hutchinson, the able resident surgeon of the institution:—

Of the 39 cases, 4 died of apoplexy between birth and 40; 9, between 40 and 50; 6, between 50 and 60; 7, between 60 and 70; 11, between 70 and 80; 1, between 80 and 90; and 1, between 90 and 100.

Hence it would appear, that the periods of life during which fatal apoplexy is most prevalent, are between 40 and 50, and between 70 and 80.

We have now to examine in what proportion of these cases disease of the heart existed.

In 4 out of the 39, the heart was found "quite healthy." In 8 cases more, no remark is made in the journals as to its condition; it may therefore be presumed to have been healthy. This affords a total of 12 cases out of 39, in which the heart was sound: in the remaining 27, it was diseased.

Thus, taking all the ages collectively, disease of the heart accompanied fatal apoplexy in no less than 27 out of 39—*i. e.* $\frac{9}{13}$ or nearly $\frac{3}{4}$.

We will now examine at which of the above periods of life disease of the heart, in connexion with fatal apoplexy, was most prevalent.

Between *birth* and 40, disease of the heart was not found in any of the 4 fatal cases that occurred within those dates. Between 40 and 50, it occurred in 8 out of 9!—a remarkable increase. Between 50 and 60, it occurred in 4 out of 6,—a decrease. Between 60 and 70, it occurred in 3 out of 7—a further decrease; and between 70 and 80, it occurred in 10 out of 11!—another remarkable increase.

It would thus appear that the periods of life during which fatal apoplexy is most prevalent, are precisely those in which concomitant disease of the heart is of most frequent occurrence; namely, between 40 and 50, and between 70 and 80.

It was stated above, that, taking all ages together, disease of the heart occurred in 9 cases out of 13, or nearly $\frac{3}{4}$, of fatal apoplexy. Now this proportion is, I apprehend, *much* greater than is generally imagined or believed; and it sufficiently evinces the importance, in medical practice, of carefully studying how far the state of the heart and that of the brain, may be allied as cause and effect.

But, in the two apoplectic periods of life, if I may be allowed the expression, viz. between 40 and 50 and between 70 and 80, the proportion is much greater; for, instead of being 9 out of 13, it is in the proportion of 9 out of 10, and 10 out of 11. Hence, it is desirable to direct our attention, in the treatment of apoplexy, to these two periods more especially; and, in order to do it with effect, it is necessary to investigate the reasons why fatal apoplexy occurs in connexion with disease of the heart, during these periods in particular.

Now, on examining the cases occurring between the ages of 70 and 80, 7 out of 10 present ossification of the heart. On the other hand, between 40 and 50, disease of the *muscular* structure of various kinds, prevailed, while ossification was comparatively rare.

Hence it appears deducible, as a generalization, that it is disease of the *muscular* structure more especially, which causes apoplexy in the earlier period of its prevalence; and that it is mainly ossification whch occasions it in the more advanced period.

It will now be asked, *why* disease of the muscular structure occasions fatal apoplexy between the ages of 40 and 50 *in particular*. To this question, the *history* of disease of the muscular structure affords a reply. Such disease is not, in general, expeditiously fatal. It usually commences insidiously, and steals on gradually—often subsisting from ten to twenty years, or more, before it produces its fatal effects. If, then, we consider that it is principally between the ages of 25 and 40 that the *causes* of disease of the muscular structure are brought into operation; if we reflect that this is the period when intellectual exertions are the most intense and sustained,—when the exciting and depressing passions have the strongest and most permanent hold,—when the physical system is subject to the greatest variety, and severity, and continuity of efforts; all of which causes, by preternaturally stimulating the heart, predispose it to muscular disease; if we finally reflect that it is before 40 that rheumatic inflammation of the heart—that fertile source of hypertrophy, &c. is most prevalent, we shall not be surprised that the seeds of destruction, sown during this period, should yield their fruits during the subsequent period; namely, between 40 and 50.

It will next be asked, why ossification causes fatal apoplexy between the ages of 70 and 80 in particular.

It is not until towards the age of 60 that the ossific tendency which characterizes old age, comes very decidedly into operation. Between the ages of 60 and 70, it makes silent progress in the heart, and between 70 and 80 it produces its fatal effects; these effects being, no doubt, assisted by disease of the cerebral arteries, which also accompanies the progress of decay.

An able writer, (in the Med. Gaz. December 12, 1835, p. 412: also, in a paper read to the College of Physicians,) who thinks that hypertrophy and apoplexy are connected by nothing more than mere coincidence, and that "the true explanation of the hæmorrhage in the brain, is to be found in the diseased state of the cerebral arteries," has applied this explanation to the above 39 cases. Too much importance certainly cannot be attached to the effect of diseased cerebral arteries in producing apoplexy; and, in order to show that I had not overlooked it, as the above writer states, I may here be permitted to reproduce a passage written five years previous to his strictures, in the first edition of this work (p. 160). "In the arteries at the base of the brain, calcareous and other degenerations are remarkably frequent, and are a principal cause of rupture of the vessels and apoplectic effusion. It is rare, indeed, to meet with instances of such effusion, exclusive of those from external violence, in which some disease of these arteries may not be detected; and it is remarkable that the disease of the artery *is in general connected with hypertrophy of the left ventricle*: whence it appears to be a result of overdistention, to which the cerebral arteries are more obnoxious than any others, in consequence of their being destitute of a cellular coat, and also of being ill supported by the pulpy yielding substance of the brain."*

But the argument of the writer in question is illogical: for, if diseased cerebral arteries can produce apoplexy while there is a *natural* state of the circulation through the heart, they will, *a fortiori*, produce it when there is either a preternaturally strong, or an obstructed, circulation through the organ, since both the one and the other tend to congest and strain the cerebral vessels. The truth manifestly is, that the cardiac and the cerebral diseases are each separately capable of giving rise to apoplexy; and that, when they co-exist, the effect is produced in a higher degree.

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* It is satisfactory to see different observers come to the same conclusions. Thus, M. Bouillaud, not aware of the above passage, says, in 1835, "I shall notice a circumstance *hitherto neglected by observers*, namely, the frequency of cretaceous degeneration, and therefore fragility, of the cerebral arteries, in subjects who die of cerebral hæmorrhage and *who are affected with hypertrophy of the left ventricle* of the heart" (Traité, ii. p. 451). The notice of hypertrophy is the more remarkable, as this writer almost invariably ascribes arterial diseases to inflammation, and not to overdistention, an idea which originated, I believe, with myself.

The same writer has adduced a case of hypertrophy of the left ventricle with "*an unusually small cavity*, and with a *contraction of the mitral valve*, through which nothing larger than one's thumb could easily pass," as "directly opposed to the opinion generally prevalent that hypertrophy is a physical cause of apoplexy." For he contends that so small a ventricle would propel a diminished, instead of an increased quantity of blood into the brain: consequently, that the apoplexy was occasioned, not by the hypertrophy, but by disease of the cerebral arteries. This case, however, proves nothing against the doctrine, that hypertrophy is a cause of apoplexy; for it is, unluckily, a case of exception, as above described at p. 255, and which, as being an exception, I have excluded from the heading of the present subdivision. Hypertrophy with contraction does not produce the effects of hypertrophy, but of a valvular or other obstruction. Besides, there was, in this case, extreme contraction of the mitral valve, which would neutralise the special effects of any form of hypertrophy! The case is no less unsuitable for establishing the writer's own doctrine, viz. that disease of the cerebral arteries is the true cause of hæmorrhage in the brain, than it is for subverting that of his opponents; for it will not be denied that a mitral valve contracted to the size of a thumb, constitutes one of the most serious obstacles to the return of the venous blood from the whole system: consequently, the brain must have been congested, and such congestion would powerfully co-operate with disease of the cerebral arteries in producing the rupture of those vessels.

As the opinions of the writer in question justly carry much weight, it has been the more necessary to point out the unsoundness of his reasonings in the present instance, in order to prevent the dissemination of a pernicious error.

Such are the grounds on which I believe that not only hypertrophy, but all kinds of obstructions to the circulation through the heart, contribute to the production of congestion, either active or passive, and of apoplexy.

Nor is it to apoplexy alone, but, on the same principle, to cerebral inflammations and irritations of every description, and even to inflammatory action in general, that hypertrophy of the left ventricle gives a tendency. The history of individuals affected with it, not unfrequently presents a striking narrative of violent head-aches, brain-fevers, various inflammatory complaints, and states of great nervous irritability and excitation. This remark has, I understand, been corroborated by the recent researches of Dr. Clendinning in the St. Marylebone Infirmary. As the opthalmic artery is derived from the carotid within the cranium, the eye participates with the brain in the effects of hypertrophy, and is vascular, brilliant, and prone to ophthalmia. The wasting away of the eye which Professor Testa has remarked as one of the effects of disease of the heart, is, with good reason, supposed by M.M. Bertin and Bouillaud to be connected with ossification of the ophthalmic arteries.

The shock of an hypertrophous left ventricle may, to a certain extent, be intercepted, and its effects on the brain counteracted, by contraction of the aortic orifice. A patient was under the care of Mr. Babington, at St. George's Hospital, September 16, 1829, for a surgical complaint, in whom the walls of the left ventricle were an inch thick, without any change of the cavity; and the aortic and mitral orifices were respectively encircled by a ring of bone as thick as a writing quill. The two valves, though overspread with calcareous scales, were capable of discharging their functions (See Fig. 15). Notwithstanding this extraordinary state of disease, the patient had attained the age of eighty without manifesting symptoms of diseased heart sufficient to arrest his own attention, or that of his medical attendants. His advanced age, indeed, proves that they could not have existed in any considerable degree. In this case, therefore, the valvular contraction appears to have been exactly sufficient to countervail the hypertrophy, and maintain the circulation in a state of equilibrium. The generality of authors, however, have greatly over-rated the power of contraction of the aortic orifice to counteract the effects of hypertrophy on the brain. They have supposed that a moderate, and even a slight degree of contraction, is sufficient for the purpose. There can be no greater error; and it is one into which they could not have fallen, had they been aware that such a degree of contraction has very little effect in diminishing the strength, tension, and regularity of the pulse. To this subject I shall revert in the chapter on valvular disease.

In the first edition, I quoted a passage from Bertin and Bouillaud, stating, that, as hypertrophy of the left ventricle caused cerebral hæmorrhage, so hypertrophy of the right caused active pulmonary hæmorrhage. I have not, however, subsequently found this coincidence to be sufficiently frequent to authorize its adoption as a general rule. I have not, in fact, been able to meet with a single case in which I could refer the hæmoptysis to hypertrophy of the right ventricle exclusively, though I have seen several in which it was connected with hypertrophy of both. Dr. Watson and M. Bouillaud have made similar observations, and the latter gentleman, in his more recent Treatise, has withdrawn the passage from his text, and placed it, as questionable, in a note. He asks whether the rarity of pulmonary hæmorrhage is not *partly* attributable to the absence of disease of the pulmonary artery. I should think that this question may safely be answered in the affirmative. Further causes may be, that the right ventricle is seldom hypertrophous to a considerable amount without contraction of its cavity, by which the quantity of blood expelled into the lungs is diminished; also, that when the pulmonary vessels are gorged, an effort is immediately made by increased respiration to relieve them,—a relief which is not enjoyed by the cerebral vessels.

SECTION V.

SIGNS AND DIAGNOSIS OF HYPERTROPHY.

THE signs of hypertrophy are of two classes: the first, called *general*, consists of its effects on the functions of the economy at large; and the *rationale* of these signs is fully explained in the preceding section: the second, for which *physical* is the most appropriate designation, comprises the impulse and sounds of the heart and the resonance of the præcordial region on percussion.

According to my experience, neither of these classes of signs, taken separately, is sufficient to indicate disease of the heart, in all cases, with complete certainty: taken conjointly, they render the diagnosis so easy, that a material error can scarcely be committed. They should never, therefore, be dissociated. At the same time, truth requires the admission that a rude, general diagnosis of marked cases in the advanced stage, (*but of no others,*) can be made by general signs alone, as was done before the discovery of auscultation: also, that many cases of hypertrophy, and nearly all of valvular diseases when yielding distinct murmurs, admit of a positive and precise diagnosis, even in the earliest stages, by physical signs alone.

General Signs.—As a systematic arrangement of signs facilitates their registration in the memory and their employment in the process of catechising a patient, it may not be irrelevant to state that, in describing those of hypertrophy, I shall follow the course of the circulation; commencing, after having noticed the action of the heart, with the circulation through the lungs, proceeding to that through the aortic system, and concluding with that through the veins.

The description of symptoms which I am about to offer, refers, it must be distinctly understood, to *simple* hypertrophy, (without valvular or vascular disease,) when it is not otherwise stated: the symptoms of hypertrophy with dilatation, which will be glanced at incidentally, are only an aggravated degree of the same,—as the reader will sufficiently understand, if duly acquainted with the foregoing principles relative to the formation and effects of these diseases. When the dilatation predominates over the hypertrophy, the symptoms, of course, approximate more nearly to those of dilatation (vid. Dilatation). The symptoms of hypertrophy with contraction will also be noticed incidentally with those of simple hypertrophy; but it may here be repeated, in general terms, that, when the contraction is considerable, it constitutes an obstruction to the circulation tantamount to a valvular disease, as explained at p. 255.

1. *Palpitation.*—By this is to be understood, a morbidly increased action of the heart both as to strength and frequency. As the hypertrophous heart acts with an energy which, even in its tranquil state, verges on palpitation, and which, under the slightest excitement, actually amounts to it, the patient experiences the consciousness of his "heart beating," more uninter-

mittingly than in any other disease of the organ. It is aggravated by stimulants of any description: as efforts, particularly that of ascending; mental emotion; flatulence; acidity or bile; spirituous or highly seasoned ingesta, and sometimes by a full meal of any kind. The violence of the attack, in the early stages, generally subsides promptly after the operation of the exciting cause has been suspended, and little remains but a slight sense of pulsation in the præcordial region. Many, especially of the working classes, become so accustomed to this, that, from unconsciousness, they deny its existence. The practitioner, therefore, should never trust to their report, but explore for himself with the hand and stethoscope. In the advanced stage, however, of hypertrophy, and still more of this conjoined with dilatation, when the capillary circulation has become embarrassed, the paroxysms of palpitation are very severe and prolonged, though they never attain that fearful extreme of violence and obstinacy which is witnessed in cases complicated with valvular, or aortic disease, or adhesion of the pericardium.

2. *Dyspnœa.*—While the enlargement of the heart is moderate, and before dropsy has supervened, the patient, during a tranquil state of the circulation, feels little or no difficulty of respiration; but he is incapable of making the same corporeal efforts as other persons without losing breath: to use a common phrase, he is "short-winded." After a respite of a few minutes, however, he recovers, and is, therefore, seldom deterred by this symptom from prosecuting his accustomed avocations. Many, indeed, become so habituated to a slight degree of dyspnœa that they deny its existence, even after ascending a staircase. The practitioner should always judge for himself by counting the pulse and respirations after a muscular effort, and ascertaining whether they are accelerated beyond the natural degree.

I have frequently observed that an individual who pants on first setting out on a walk, is capable of sustaining great exertions without inconvenience when he gets warm, and the blood is freely determined to the surface.

When the disease has proceeded so far as to occasion external dropsy, and sero-sanguineous congestion of the lungs, more or less dyspnœa becomes almost habitual, and it sometimes occurs, conjoined with palpitation, in paroxysms of excessive severity, especially in asthmatic subjects. From this period, indeed, the symptoms are a compound of those of hypertrophy and those of an obstructed circulation, the latter of which are more particularly considered in the article Dilatation. The cause of obstruction has been explained at p. 253.

3. *Cough.*—There is generally little or no cough in the early stages, but it occasionally supervenes when dropsy appears, in connexion with which, more or less sanguineous and serous congestion almost invariably takes place in the lungs, and gives rise to the symptoms in question. The cough is seldom considerable unless the patient is subject to chronic bronchitis, either in the dry or pituitary form.

4. *Hæmoptysis.*—This is the result of a too impetuous discharge of blood into the capillary system. It is of rare occurrence, for the reasons assigned above (p. 262).

5. *Pulse.*—The pulse in hypertrophy of the left ventricle undergoes, from valvular and other lesions, a variety of modifications which disguise its real nature. It must, therefore, be studied in cases totally exempt from complication. In such, it is almost invariably regular, and bears strict relations in strength and size to the thickness and capacity of the left ventricle. Thus, in simple hypertrophy, it is stronger, fuller, and more tense than natural: it swells gradually and powerfully, expands largely, *dwells long under the finger*, and, in anæmic subjects, (but no others,) is sometimes accompanied with a thrill or vibration. These characters are still more marked in hypertrophy with dilatation, so long as the hypertrophy is predominant; but when the dilatation has proceeded so far as to diminish the contractile power of the muscular fibres, the pulse, though still full and sustained, is soft and compressible. In hypertrophy with contraction of the cavity, it is tense, but small, expanding little under the finger; and, if the contraction be great, it loses its tension and becomes weak as well as small, from the insufficient quantity of blood propelled into the arteries.

The strength, largeness, and tense prolongation of the pulse of hypertrophy with dilatation, are often so remarkable, that, from this sign alone, the practitioner may often make a successful conjecture at the nature of the disease; for inflammation only can impart similar strength, and comatose affections, similar prolongation.

The pulses of hypertrophy and hypertrophy with dilatation now described, may present exceptions; for depression or exhaustion of the nervous system, whether from the advanced stage of the disease, or from accidental, debilitating causes of any kind, may so neutralize the contractile energy of the heart as to enfeeble the pulse. Thus, according to my observation, it is an ordinary occurrence for a pulse, which was large, strong, and regular in the early stages, to become more or less small, weak, and even irregular before death. I have also repeatedly noticed that a hypertrophic pulse has become permanently small and weak from the date of an apoplectic or paralytic attack, which has debilitated the general system, and even put a period to previous headaches. The pulse may also become temporarily small and weak during severe attacks of palpitation and dyspnœa, by which the heart is gorged and rendered incapable of freely expelling its contents. I have observed the same to result from great phlethora, the pulse becoming full and strong after moderate bleeding. These exceptions, being referable to obvious causes, confirm the general rule.*

6. *Affections of the Head.*—These exist in a large proportion, but not in all. The patient complains of a "rushing of blood to the head" on making any corporeal effort or stooping; of more or less intense throbbing and lancinating headaches, aggravated by the recumbent position, and especially by the act either of suddenly lying down or rising up; of vertigo, tinnitus aurium, scintillations and other visual illusions; and

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* Dr. Graves found, in five or six cases of "very great hypertrophy with dilatation," that the pulse was not accelerated, (after the first quarter of a minute,) by substituting the erect or sitting, for the horizontal position, as it is in health, and, still more, in all diseases of debility,—the change amounting to from 6 to 15 beats per minute in the healthy, and from 30 to 50 in the debilitated. "It would be premature," says he, "to inquire into the cause of this phenomenon, but it immediately suggests itself to the mind, that it depends on the increased strength and energy of the left ventricle when in a state of hypertrophy, and which, in a great measure, place its contractions, as it were, beyond the influence of those causes which, in other diseases, attended with debility, and even in many persons in health, enable a change of posture to produce so remarkable an alteration in the frequency of the pulse" (Dub. Hosp. Rep. vol. v. p. 567). I imagine that the cause of the frequency is simply, the greater hydrostatic pressure on the heart in the erect position.

sometimes of a lethargic somnolency, which so completely subdues the faculties both of the mind and the body, as utterly to incapacitate him for every species of exertion. These symptoms, if not relieved, are apt to terminate in palsy, apoplexy, or inflammation of the brain. From these catastrophes the patient is occasionally preserved by the opportune occurrence of epistaxis, to which, happily, he has an increased predisposition. From the circulation in the early stages of hypertrophy being active in the eye, this organ is often bright and sparkling, and sometimes vascular or blood-shot.

7. *Complexion.*—The effect of hypertrophy is to heighten the colour so long as the capillary circulation continues unembarrassed, but afterwards to diminish and change it. Every individual, however, does not acquire a florid colour. Whether he acquires it or not, depends, in fact, upon his original complexion, the series of changes being different in those who are naturally florid, and those who are pale. In the former, the colour becomes remarkably vivid, and, being generally accompanied with plethoric turgescence, it gives the aspect of health and good condition. But when the capillary circulation begins to labour, the red changes into a purplish patch on the cheeks; the nose and lips become more or less purple, violet, or livid, and the intermediate skin becomes pale and sallow. In great hypertrophy with dilatation, the purple and violet colours are sometimes of the deepest dye. In those, on the contrary, who are naturally devoid of colour, hypertrophy either does not excite it at all, or merely increases, in a slight degree, the *general* vascularity of the face. This vanishes entirely when the capillaries become obstructed, and is superseded by universal cadaverous paleness and sallowness, extending sometimes even to the lips. They, however, are generally somewhat livid. These distinctions have been wholly overlooked by authors, who have created much confusion by assigning a red face to all hypertrophic subjects without distinction.

8. *Serous Infiltration.*—This, for reasons already assigned, (p. 254,) seldom appears before the hypertrophy is very considerable, or becomes conjoined with enfeebling dilatation. It occasionally shows itself first in the face: a circumstance attributable to the great number and size of the cerebral arteries, and to the force

with which the blood is injected into them, in consequence of their proximity to the heart. More commonly, however, it begins in the ankles, and gradually becomes universal. With dropsy, supervene, in a greater or less degree, all the other symptoms of an obstructed circulation.

9. *Angina Cordis.*—In a great number of cases of hypertrophy, I have found patients complain of a dull, though severe aching pain in the region of the heart, usually extending towards the shoulder and down the inside of the arm to the elbow or below. It is generally aggravated by exertion, especially walking up-hill or against the wind. It appears to me to be dependent upon overtension of the heart, as I have generally found it cease or greatly diminish after one or two abstractions of ℥vi or viii of blood, and a few purgatives. Angina, however, is not an *essential* symptom either of hypertrophy, or of any other disease of the heart, though there is no form which I have not known it to accompany. Slight degrees are perfectly common in nervous and hysterical subjects, wholly exempt from organic disease. The old writers erroneously supposed it to be restricted to ossifications. It is true that, in these, it is apt to attain its highest degree of agonising intensity.

Signs of Hypertrophy of the Right Ventricle.—Hypertrophy of the right ventricle produces, according to Corvisart, a greater difficulty of respiration, and a deeper colour of the face, than is produced by the same affection in the left ventricle. I have not been able to verify this. Corvisart was possibly mistaken, from his imperfect acquaintance with valvular disease, especially mitral regurgitation, which probably occasioned, not only the dyspnœa and deep colour, but the hypertrophy itself, as explained above at p. 249. Another alleged sign is, the more frequent expectoration of pure arterial blood. This is very questionable.

The only signs of value besides the physical, (viz. increased impulse and dulness on percussion under the lower portion of the sternum,) are, 1. absence of the strong, large, and prolonged pulse of hypertrophy of the left ventricle, in the few cases in which the right alone is hypertrophous: 2. turgescence of the external jugular veins accompanied by pulsation synchronous with that of the arteries. This was broached by Lancisi as a sign of "aneurism," *i. e.* hypertrophy with dilatation, of the right ventricle.

Though rejected by Corvisart,—in my opinion on insufficient grounds, it is approved of by Laennec, who found it to exist in every case of rather considerable hypertrophy of the right ventricle, and never in that of the left unless the right was simultaneously affected (Laennec de l'Auscult. tom. ii. p. 505). I have rarely known it to be absent in cases where dilatation was conjoined with hypertrophy of the right ventricle. Of such cases, therefore, I regard it as one of the best general signs, though, after all, it is but an equivocal one.

The explanation of the phenomenon offered by M. M. Bertin and Bouillaud, and by the latter in his subsequent work, (ii. p. 449,) appears unsatisfactory. "The jugular or venous pulse," says he, "is seen in those cases only in which dilatation accompanies hypertrophy, and in which the auriculo-ventricular orifice, being greatly enlarged, is no longer completely closed by its valve: thence ensues a regurgitation of blood into the great veins during the contraction of the right ventricle." Hypertrophy with dilatation has certainly, though not always, the effect of enlarging the auriculo-ventricular orifice; but the valve in most instances expands in a corresponding degree; as I have repeatedly found. I apprehend, therefore, that the venous pulsation, in the cases where I have observed it to exist, was not attributable to regurgitation: in substantiation of which opinion I may say, that regurgitation would be attended with a bellows or other such sound: this sound, however, is not found to be a concomitant of jugular pulsation. Is the rationale of the phenomenon as follows? namely, as the ventricle, when hypertrophous, contracts with augmented power, the recoil of the tricuspid valve is preternaturally impetuous: hence, the column of blood in the act of descending into the ventricle, is repelled with such an increase of force, that its impulse is propagated as far back as the jugular veins. This effect will be more considerable when the orifice and valve are enlarged, because the quantity of fluid repelled will be greater. The effect will also be favoured by congestion of the great veins, (a state which generally accompanies hypertrophy with dilatation of the right ventricle,) because, when congested, they are more tense, unyielding tubes, and more readily transmit an impulse.

But the jugular pulsation is double: a weaker pulsation precedes that occasioned by the ventricular systole. The weaker is occasioned by the auricular systole, and the mechanism of its formation I conceive to be this: at the time that the auricle contracts, the ventricle is in a state of moderate or natural fulness: it therefore offers a certain degree of resistance to the ingress of more blood from the auricle; consequently, so much of the blood compressed by the auricular systole as cannot get forward into the ventricle, is forced back into the veins and causes their pulsation. Some contend that the auricle occasions no jugular pulsation, founding their opinion on the assumption that the ventricle is empty at the moment that the auricle contracts, and that, therefore, the whole of the auricular blood must descend into the ventricle. Such an assumption, however, according to the evidence adduced in the first part of this work, (p. 16, 21, and 61,) is incorrect.

A difficulty has sometimes been experienced in distinguishing jugular pulsation from that of the carotid arteries. Error may easily be avoided by observing that the jugular pulsation is confined to the lower part of the neck, and is far on the humeral side of the carotid. The pulsations of this artery, on the contrary, extend as high as the angle of the jaw, and in the direction of the anterior margin of the sterno-cleido mastoideus muscle.

The jugular turgescence, moreover, disappears in some degree during inspiration and reappears on expiration: which movements, therefore, must not be confounded with the pulsations answering to the systole of the ventricle.

General Signs of Hypertrophy of the Auricles.

There are none that are distinguishable from those of disease or obstruction in the corresponding ventricle or orifice, to which the hypertrophy of the auricles owes its origin. The detection of hypertrophy of the auricles is of little importance, as it is the cause that produced it, which is the source of danger.

*Physical Signs of Hypertrophy.**

Impulse.—According to Laennec, the impulse is best appreciated by the ear applied to the stethoscope. I participate in this opinion; for I continually meet with cases in which an application of the hand would not authorise an assertion that there was an increase of impulse, yet an application of the stethoscope renders that increase distinctly appreciable. It was, perhaps, to these cases of slight increase of impulse that Laennec alluded, when he said that the application of the hand was a very fallacious mode of appreciating the impulse. This mode, however, is far from useless in examining great degrees of impulse. It is my own invariable habit to begin an examination by application of the hand; whence I acquire a general idea of the extent and strength of the impulse, and a knowledge of the precise spot where it is strongest, and where, consequently, it is best to apply the stethoscope. A good idea of the heaving nature of the impulse, is acquired by watching the rise and fall either of one's own hand applied to the part, or the head of an explorer resting on the stethoscope. The *immediate* application of the ear is, in my opinion, the least delicate mode of estimating the impulse, as slight degrees are not perceptible by it, and, in high degrees, it is unnecessary, as then, even the hand alone will generally answer every purpose. However, M. Bouillaud says, " in a good number of cases, (I do not say in all,) the immediate application of the ear is preferable to the use of the cylinder in appreciating the shock of the heart" (Traité, i. 140, note). I do not understand to what class of cases he alludes.

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* For the rationale of the impulse and sounds in the several varieties of hypertrophy, the reader is referred to p. 67.

In *simple hypertrophy*, " the impulse," says Laennec, " communicated by the stethoscope while the patient is in a calm state, is usually so strong as distinctly to raise the head of the observer, and sometimes even sufficient to produce a shock disagreeable to the ear. The greater the hypertrophy, the longer this heaving takes for its performance. When the malady exists in a great degree, we evidently perceive that the heaving takes place with a gradual progression; it seems as though the heart swelled and applied itself to the parietes of the chest, at first by a single point, then by its whole surface, and finally sank back in a sudden manner." This sinking back did not sufficiently arrest the attention of Laennec. In the first edition of this work, I called attention to it, as a new sign of hypertrophy, under the name of the *back-stroke:* but the term *diastolic impulse*, which I now propose to use, is a more descriptive appellation. It is occasioned by the diastole of the ventricles, during which action the heart sinks back from the walls of the chest, and this sinking back terminates in a jog or shock, occasioned by the refilling of the ventricles, and constituting the diastolic impulse in question. It is stronger, cæteris paribus, in proportion as the heart is thicker and more capacious. Accordingly, I have found it strongest in hypertrophy with dilatation, but it may also be very considerable in simple hypertrophy. In the healthy heart it is not perceptible, neither is it in dilatation without hypertrophy.*

A strong, slowly heaving impulse, then, is the principal sign of simple hypertrophy; and the affection may be known to be greater, when the impulse is followed by a diastolic impulse. Both these signs exist in hypertrophy with contraction, but in a less degree, and the diastolic impulse may be absent if the hypertrophy is not great.

In simple hypertrophy and that with contraction, the impulse is seldom perceptible much beyond the præcordial region, except during attacks of palpitation.

In estimating the impulse in this and every other form of disease, it is to be taken into account that, other circumstances being equal, the impulse is more perceptible in proportion as the walls of the chest are thinner. Thus, it is the most distinct in the emaciated, and in children; whereas, in very stout and muscular subjects, it may be barely perceptible.

In hypertrophy with dilatation, the signs are a compound of those of hypertrophy and those of dilatation. The contraction of the ventricles can easily be felt by the hand applied to the præcordial region, and we find, especially during palpitation, smart, violent shocks, which strongly repel the hand. In extreme cases, I have known the extent of these almost equal that of the expanded hand. If we attentively examine the patient when most calm, we see that not only his whole chest and the pit of the stomach, but his head, his limbs and even the bed-clothes, are strongly shaken at each contraction of the heart. The pulsations of the carotids, the radials, and the other superficial arteries, are often visible. The impulse of the heart can sometimes be distinctly felt as high as the clavicle on the left side of the thorax, and sometimes even on the left side of the back, especially in meagre subjects and children.

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* M. Bouillaud thinks that the *back-shock* or *diastolic impulse* was *new*, (non moins *neuf* qu'intéressant,) when he wrote on it (Traité, i. p. 148). In this idea, he does not, I think, do full justice even to M. Laennec. "M. Laennec," says he, "teaches that the impulse of the heart is only perceptible at the moment of the ventricular systole: consequently, that it is *unique*, *simple*, and not *double*." But Laennec applies the expression "s'affaisse *tout-a-coup*" to the diastole in hypertrophy; whence it appears to me that he was not wholly a stranger to the diastolic impulse. But though Laennec only glanced at the phenomenon, it was fully described by myself, as seen above, several years before M. Bouillaud published. I must entirely dissent from him when he adds, "a phenomenon still more curious, is, that for one systolic impulse there may be two diastolic impulses," (p. 147,) the first or systolic impulse only being accompanied with a radial pulse. He cites a case in exemplification. But I have already shown (p. 64) that he has mistaken the entire subject. These intermediate impulses without pulse, are not diastoles, but systoles, of the ventricles, as proved by their being invariably attended with a first sound or click of the auricular valves, and often with a barely perceptible pulse. This is only an offset of the same error, which led him to ascribe the intermediate sounds to auricular contractions.

In *hypertrophy with a predominance of dilatation*, the impulse is ordinarily not considerable; but it becomes very marked during palpitation, especially if accompanied with fever, and it has a very different character from that occasioned by simple hypertrophy. The beats, as well described by Laennec, are strong, hard, and produce a shock analogous to the blow of a hammer; but the blow seems to strike a small space, it expends itself, as it were, on the thoracic parietes, and does not communicate to the head a heaving proportioned to its force: it differs, in short, from the impulse occasioned by great hypertrophy, in the circumstance that, in the latter, the ventricles in a distended state, seem to heave with their whole length against the thoracic parietes, which yield to the effort; while, in the former case, the point only of the heart seems to strike the parietes with a sharp, smart, accurately circumscribed blow, only capable of producing a sort of concussion, rather than a real heaving.

When the impulse is increased on one side only of the præcordial region, that is, under the inferior part of the sternum, for the right side, and between the cartilages of the fifth and seventh left ribs, for the left, we infer that the corresponding ventricle only is affected: and when it is increased on both sides, we conclude that both are affected, which is the more common case.

In hypertrophy, and hypertrophy with dilatation, free from valvular disease, the beats of the heart, even during palpitation, are rarely irregular in the early stages of the disease, while the patient's general strength continues little impaired; but I have often met with temporary irregularity during excessive dyspnœa, and with permanent intermission when the strength and vital powers failed in the late stages, especially on the approach of dissolution. Nervous and dyspeptic intermission may, of course, affect a patient labouring under hypertrophy; but this is accidental, and not a part of the disease.

The impulse of the heart is diminished by loss of blood, diarrhœa, any exhausting disease, rigid and long-continued abstinence, and, in general, by all the causes capable of producing debility. Consequently, a moderate hypertrophy might, without due care, be overlooked in a patient under any of these circumstances. It has frequently occurred to me to notice, that patients, cured of hypertrophy by tranquillising means, have eventually disbelieved that they had ever laboured under it—especially when biassed by the opinion of others.

The impulse of the heart, moreover, occasionally ceases entirely, or becomes a mere oppressed struggle, (even in cases of very marked hypertrophy,) when there supervenes intense dyspnœa referable to some affection of the lungs, especially peripneumony, pleurisy, œdema of the lungs, asthma, and the congestions which form during the last moments of life. The sounds likewise diminish: no inferences, therefore, should be drawn from an exploration made under such circumstances.

Sounds.—Hypertrophy has the effect of deadening the sounds of the heart. In *simple hypertrophy*, the *first* sound, *i.e.* that produced by the ventricular contraction, is duller and more prolonged than natural, in proportion as the hypertrophy is more considerable. When the hypertrophy exists in an extreme degree, the first sound becomes nearly extinct: Laennec says, wholly; but I have never found it so. It may always, I think, be heard by placing the stethoscope on that part of the ventricles which is in contact with the walls; namely, about the apex. The *second* sound, *i. e.* that produced by the sigmoid valves during the ventricular diastole, is very feeble; in extreme cases, says Laennec, it is scarcely perceptible; but I have always found it distinct immediately over the sigmoid valves, and thence up the courses of the aorta and pulmonary artery. The interval of repose is shorter than natural, in consequence of the first sound being longer. Both sounds are proportionably weaker in *hypertrophy* with *contraction*. In most cases of both these forms of hypertrophy, the first sound can scarcely be heard under the left clavicle and at the upper part of the sternum, but the second generally can.

Each sound of the heart, though essentially one, consists of the sounds of the two sides united. This is proved by a bellows-murmur in the left præcordial region being audible in the right, and *vice versâ*. It does not follow, therefore, that when one ventricle only is hypertrophous, the sound of the heart in general should be very limited in its range; for that of the other ventricle will be heard over an extent proportioned to its intensity, though not quite so far as when strengthened by its fellow. On the other hand, a morbidly increased sound of one ventricle, as by dilatation or a bellows-murmur, will be heard *alone* at points beyond the range of the natural sound of the other or healthy ventricle. Accordingly, it is only in hypertrophy of both ventricles that we must expect to find the sounds confined within very narrow limits.

The second sound is more audible than the first from the semilunar valves, up the sternum, to the clavicles. This remark applies both to hypertrophy and to the healthy state. The reason is, that, as the sound is created by the semilunar valves, it is transmitted along the aorta and pulmonary artery, (Exp. on the sounds, Obs. 12, p. 34,) and likewise through the sternum. For the same reason, the sound is often distinct at the clavicles when it is drowned in the præcordial region by a valvular, or pericardiac murmur, or a pulmonary râle.

In *hypertrophy with dilatation* the sounds are increased to their maximum, being louder than in any other disease of the heart, especially during palpitation. The first is, as it were, a compound of the sound of dilatation and that of hypertrophy: namely, from dilatation it derives a loud, abrupt commencement, and from hypertrophy, a prolonged termination, as explained at p. 63 and 69. The second sound, though not changed in character, is louder than natural. These sounds may frequently be heard over the whole chest both posteriorly and anteriorly, especially in children and meagre subjects.

In *hypertrophy with a predominance of dilatation*, the first sound is not so loud as in the preceding variety, nor has it a prolonged termination, but is short and smart like the second, being produced almost entirely, I conceive, by the extension of the auricular valves. The second sound is not altered, but is a degree louder than natural, from the quickness of the ventricular diastole.

The sounds of the heart in every form of hypertrophy, may be diminished by the same causes that diminish the impulse. They are specified at p. 274.

In a very few cases of great hypertrophy with dilatation, a slight, soft murmur in the aortic orifice accompanies the first sound, as already explained at p. 94. It is, I think, occasioned by the anæmia, which frequently supervenes in the last stage of this disease.

*Resonance** of the præcordial region on percussion is deficient in *simple hypertrophy*, if the heart is considerably enlarged; but, as *hypertrophy with dilatation* is the disease in which the organ attains the greatest volume, it is that in which resonance is most frequently and most extensively deficient. The line of dulness where the heart comes in contact with the walls, may be traced with great precision; and it often forms a circle of two, three, and occasionally four inches in diameter. (See Percussion, p. 5.) In all cases of considerable enlargement, the dulness as well as the impulse, are lower down than natural, except in adhesion of the pericardium; for the heart is then more or less braced up by the adhesion.

Emphysema counteracts dulness, in consequence of the lung's advancing in front of the heart. Percussion should then be made during the state of expiration, and while the patient leans forward. I have known double emphysema depress the heart completely into the epigastrium.

Prominence of the præcordial region.—This was noticed as a sign of hypertrophy by the writer, in the first edition of this work, p. 579, 467, 130, &c. M. Bouillaud has more recently observed the same: he says, "The prominence of the præcordial region had not yet been noticed, to my knowledge, by any of the authors who have published on the diseases of the heart" (Traité, ii. 444, and i. 150). It is a sign of only secondary importance, since it does not exist till the hypertrophy is very considerable, when it is sufficiently indicated by other signs.

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* See much valuable information on this subject in the "Procédé Opératoire" of M. Piorri. Paris, 1830. p. 112, et seq.

SECTION VI.

PROGRESS, TERMINATIONS AND PROGNOSIS OF HYPERTROPHY.

Progress and termination of Hypertrophy.—Hypertrophy, while moderate and not complicated with any mechanical impediment to the circulation, is productive of very little inconvenience. This is especially true with respect to children. In them, the heart is naturally larger in proportion than in adults; and in many this amounts to a very considerable degree of hypertrophy with dilatation, accompanied with greatly increased impulse and sound; yet the general symptoms manifested by such are often scarcely appreciable, and the increased action itself subsides towards the period of puberty by the establishment of a more correct proportion and equilibrium between the heart and the system.

At the adult age also, and during the whole period of manhood, an individual of an otherwise sound and vigorous constitution may be affected with hypertrophy to a moderate extent, without experiencing any sensible deterioration of the general health, (with the exception of being more liable than others to phlogistic and cerebral affections,) or any diminution of muscular force and activity: and if his habits with respect to diet and exercise be moderate, he may pass a long series of years, and even attain the extreme period of senility, without being conscious that he is the subject of organic disease. The only general signs denoting the existence of the malady, will be, perhaps, a little shortness of breath on exertion, and occasional feelings of slight palpitation. Amongst the labouring classes these symptoms, even in a considerable degree, are so little regarded, that their presence is often disavowed by the patient, though manifest to the physician. I recently saw an athletic, hard-working man, weighing, according to his own account, not less than twenty stone, with enormous hypertrophy and dilatation, who assured me that "his palpitation had quite left him for a month," yet the heart was acting with a violence that was truly astonishing.

If, however, an individual affected with hypertrophy abandon himself to intemperate living, or engage in occupations requiring great corporeal exertion, he rarely fails to bring on either apoplexy, palsy, or an aggravated state of the hypertrophy, which, if not removed by speedy and judicious treatment, embitters the remainder of his existence, as well as curtails its span.

The celerity with which these accidents are induced, depends on circumstances. In general, the progress of hypertrophy is very slow and gradual, but in some cases it is rapid: in several instances I have known it terminate fatally within a year from its commencement.

The circumstances occasioning these variations are connected with, 1. the form of the disease; 2. its complications; 3. the nature and intensity of the external exciting causes; and, 4. the constitution of the patient.

It is of the utmost importance that the practitioner be able to form some estimate of the influence of these circumstances; for it is by this means only that he can foresee the course of the disease, and direct his treatment with judgment and decision. It may be useful, therefore, to enlarge a little on this subject.

1. The progress and termination of hypertrophy are influenced by the form of the disease. *Simple hypertrophy* is more apt than any other form to induce apoplexy or palsy while the patient is apparently in perfect health. This is to be accounted for by its tendency to create plethora, while, at the same time, it does not incapacitate the patient for active corporeal exercise, and the pleasures of the table. If a premature death does not occur from apoplexy or palsy, simple hypertrophy runs a more chronic course than any other form of the disease.

Hypertrophy with dilatation, especially if great, is a far more harassing, dangerous, and, if I may be allowed the term, *acute* affection than the preceding. All its symptoms are more violent, and its course is more rapid. It is somewhat less apt to produce unexpected attacks of apoplexy; probably because the greater dyspnœa which it occasions deters the patient from violent exercise and high living. When once general dropsy appears, and shows a decided disposition to recur again and again, notwithstanding judicious treatment, the malady hurries with an uninterrupted course to its fatal termination.

2. The progress and termination of hypertrophy are influenced by its complications. When hypertrophy is connected with contraction of an orifice, regurgitation through a valve, disease of the ascending aorta or arch, or any other material obstacle to the course of the blood, the symptoms are greatly aggravated. For, in the first place, in consequence of that obstacle, the hypertrophy proceeds to a greater extent; and, secondly, the violent struggles of the heart to surmount the obstacle, subvert the general balance of the circulation. To speak more explicitly, suppose the obstacle to be situated in the aortic orifice. While the left ventricle is palpitating to disgorge itself through the contracted aperture, the right, acting in concert with it, deluges the lungs with an inordinate quantity of blood; whence ensues a paroxysm of dyspnœa: next, in consequence of the pressure of blood through the lungs, the supply to the left ventricle is increased: this ventricle, therefore, instead of relieving its own engorgement by palpitation, only aggravates it, and the fit does not subside until either the heart becomes gradually exhausted by its own efforts, or (what is more common) until the internal congestion is relieved by determination to the surface, or a copious discharge of watery mucus from the lungs. The most violent paroxysms of palpitation and dyspnœa that I have witnessed, have occurred in the particular complication described, that is, in hypertrophy with valvular disease. In other cases, however, there may exist a greater feeling of suffocation, as will hereafter be explained in the chapters on diseases of the valves, and on polypi.

Adhesion of the pericardium, which rarely fails to produce hypertrophy with dilatation, is an extremely formidable complication of this malady. It greatly aggravates all the symptoms, and accelerates the fatal event. It is not unusual for this to take place within the period of a year, and I have known it occur in nine months. I entertain little doubt that this rapid course of the malady is, in part, referable to the injury done to the muscular substance by inflammation propagated to it from the pericardium; for it has already been shown (p. 246) that inflammation is a cause of hypertrophy, and it will hereafter appear that it may also occasion softening. For the same reasons, valvular disease resulting from endocarditis, is in general a more serious complication of hypertrophy, than when it steals on gradually from causes independent of inflammation. It is scarcely necessary to add, that the worst cases of adhesion of the pericardium and valvular disease, are most commonly those which result from rheumatic inflammation.

Febrile or inflammatory complaints supervening upon an advanced degree of hypertrophy, exasperate the malady in a surprising manner, so as not unfrequently to carry off the patient in the course of a few days. The effect seems to be produced by the febrile excitement keeping up, as it were, a perpetual fit of palpitation and embarrassment of the circulation, which the constitution cannot support beyond a brief period. Peripneumony and extensive vesicular bronchitis have pre-eminently this effect: apparently because they not only excite the heart, but obstruct the circulation through the lungs.

3. The progress and termination of hypertrophy are influenced by the nature and intensity of the external exciting causes.

The principal of these are, over-exertion, excesses at table, and mental perturbation, the latter of which, though not strictly external, may be ranged under this head. The effect of these requires no explanation; but it may be said, that the injurious influence of over-eating and drinking is greatest in simple hypertrophy, because it generates plethora and increases the tendency to apoplexy; while over-exercise and intemperance are more prejudicial in hypertrophy with dilatation, because they increase the dilatation, which is the more dangerous part of the disease.

4. The progress and termination of hypertrophy are influenced in a remarkable degree by the constitution of the patient. The robust resist its encroachments much longer than those who are delicate and effeminate: and if the former, either from bad air and diet, from disease, or from age, become unhealthy, anæmic, emaciated, and feeble, they are rendered much more susceptible of the effects of the disease. Excessive blood letting, on Alber-

tini and Valsalva's plan, for the cure of hypertrophy, produces the same effect, and thus defeats its own object. It is for this reason that I have proposed another mode of blood-letting, &c., founded on a different principle, as will presently be explained.

Prognosis.—The general prognosis is favourable in the early, and unfavourable in the advanced stages of the disease, when dropsy has appeared and obstinately recurs. This is especially the case in the aged, and in feeble, shattered constitutions. The particular prognosis must be founded on an estimate of the various circumstances of each case, formed according to the above rules.

SECTION VII.

TREATMENT OF HYPERTROPHY.

BEFORE the introduction of auscultation, when practitioners could not distinguish disease of the heart with any certainty, and seldom before it was in an advanced stage, they generally considered it as hopeless, and contented themselves with palliating urgent symptoms. Nor can this be a subject of surprise, for, in that stage, the disease most frequently *is* hopeless so far as a cure is concerned. But, since it has become possible, by the aid of auscultation and the improved knowledge of general symptoms to which it has led, to detect not only the slighter degrees of hypertrophy or dilatation, but even the mere tendency to those affections; and since it has been fully proved that, in their early stages and sometimes even when far advanced, they are within the resources of the curative art, the practitioner would be wanting in the performance of his duty to his patient were he not to aim at effecting a radical cure, rather than content himself with merely palliating symptoms.

In the treatment, it is obvious that the first care should be, to remove any known exciting cause of the malady, as violent exercise, intemperance, mental excitement, &c. It is equally obvious that, as this malady consists in an increased power and action of the heart, blood-letting and other reducing and tranquillizing means are the appropriate remedies. Laennec strongly recommends that they be employed, with courage and perseverance, on the plan of Albertini and Valsalva. I cannot say that my own observation leads me in the least degree to coincide with him in this opinion. I shall first, therefore, give a sketch of the treatment alluded to, as the sanction accorded to it by names of the highest authority renders it at least deserving of attentive consideration: and I shall afterwards point out in what respects it appears to me to be objectionable.

This treatment, according to M. Laennec, ought to be prosecuted in an energetic manner, especially at the commencement; and, in aiming to enfeeble the patient, we ought, says he, much more to fear resting short of the mark, than exceeding it. We should commence by abstracting blood as copiously as the patient can support without falling into a state of sinking, and we should repeat the operation every two, four, or eight days, until the palpitation has ceased, and the heart no longer gives, under the stethoscope, more than a moderate impulse. We should, at the same time, reduce to at least one half, the quantity of aliments which the patient ordinarily takes, and diminish even this quantity, if he preserve more muscular strength than suffices to take, step by step, a walk of a few minutes in the garden. In a stout adult, Laennec usually reduces the quantity to fourteen ounces a day, amongst which he thinks there should be only two ounces of white animal food. If the patient wish to take broth or milk, he counts four ounces of these liquids for one of animal food. Wine ought to be interdicted. When the patient has been about two months without experiencing palpitation, and without strong impulse of the heart, we may dispense with the bleedings, and somewhat diminish the severity of the regimen, if habit has not yet been able in any degree to reconcile the patient to it. But it is necessary to revert to the same means, and with equal rigour, if in the sequel the impulse of the heart increase again. We ought not to have confidence in the cure until the expiration of a year of complete absence of all the symptoms, and especially of all the physical signs, of hypertrophy. We must be afraid, pursues Laennec, of allowing ourselves to be deceived by the perfect calm which blood-letting and abstinence sometimes very promptly produce, especially if we have commenced the treatment at a period when the hypertrophy was already accompanied with extreme dyspnœa, with anasarca, and with other symptoms which gave reason to fear an approaching death.

If we begin the treatment of hypertrophy of the heart at a period when it has already produced severe effects, particularly anasarca, ascites, œdema of the lungs, and a very marked state of cachexy, we ought not on that account to shrink from bleeding and abstinence.

To obtain success by the treatment described, it is necessary, according to the same author, that the physician and the patient arm themselves with almost equal patience and firmness; for it is not more difficult for the latter to resign himself to a perpetual fast and frequent blood-lettings, than for the former to struggle daily against the opposition of relations, friends, and the discouragements which cannot fail to seize upon the patient in a treatment which ought to continue *at least several months, and sometimes to be prolonged during several consecutive years!*

Such is the manner in which M. Laennec employs the treatment of Albertini and Valsalva, and he states that he could cite a dozen instances of cures of hypertrophy, either simple or with dilatation, which have not been falsified for several years. One important case, which he details, seems to prove that the treatment causes atrophy of the heart; for the organ was remarkably less than the fist of the subject, and was shrivelled or wrinkled in a longitudinal direction.

My objections to the treatment described are founded on the circumstance that, though I have invariably found the greatest benefit to be derived, in the early stages, from sparing abstractions of blood at intervals of two or three weeks or more, I have constantly noticed that when, from the severity of the dyspnœa and palpitation in the advanced stages of the complaint, the practitioner was induced, or *thought himself compelled, to resort to frequent bleedings at short intervals, the patient, though perhaps temporarily relieved, progressively declined from that moment, dropsy increasing, and the paroxysms recurring more frequently and with greater violence, until they eventually terminated in his destruction.* Now, on comparing a patient under these circumstances with one under the influence of mere reaction from loss of blood, (of which the experiments on dogs, described at p. 100, present a graphic exemplification,) the analogy appears to me to be very intimate. In both, the violence of the heart's action, so far from being repressed by a reiteration of the blood-letting, is only increased: in both the blood is, and necessarily must be, attenuated and deteriorated, in consequence of the fibrinous portion and red globules being replaced to a far greater extent than natural by serum, which is more expeditiously regenerated; in both, in short, there exists the state of anæmia, which is invariably attended with a quick, jerking beat of the heart and arteries, palpitation and breathlessness on exertion or excitement, and that disposition to serous infiltration, which is, in popular language, called "dropsy from debility."

These, then, are the causes of the patient's decline. The hypertrophic palpitation and tendency to dropsy are aggravated by the superaddition of anæmia.

Hence it appears that the indications in the treatment of hypertrophy, are, to diminish the quantity, without materially deteriorating the quality of the blood; and to do this in such a manner as, without producing either reaction or anæmia, permanently to enfeeble the action of the heart and the energy of the circulation. These indications have seemed to me to be fulfilled in the safest and most effectual manner by the following means, the efficacy of which I have tested on a large scale since I originally proposed them in the first edition of this work.

Four, six or eight ounces of blood should be taken every two, three, four, or six weeks, according to the age and strength of the patient, so as merely to keep down palpitation, dyspnœa, and strong impulse of the heart. If the head be much affected, the blood should be drawn by cupping from the nape of the neck; but it must be clearly understood that, in case the cerebral symptoms amount to an indication of apoplexy, or of inflammation of the brain, the practitioner must not consider himself limited to the number of ounces above stated, but must bleed according to the principles which regulate the treatment of these affections.

In case of angina cordis, it might be supposed that cupping on the præcordial region would be more efficacious in relieving the pain, than bleeding from the arm; yet experience has shown me that there is scarcely a choice between the two modes, the relief seeming to result rather from the tension of the vascular system being taken off, than from the counter-irritant effect of the cupping.

In very plethoric individuals, the pulse is sometimes small and languid or *oppressed*, though there be a heaving, hypertrophic impulse of the heart. Under these circumstances, strangers to auscultation are often deterred from bleeding, by the impression that the pulse is one of debility, and that the palpitation, dyspnœa, angina, headache, &c. are nervous. The auscultator, however, may bleed under the confident assurance that the pulse will rise, and the other symptoms abate, when the tension of the vascular system is removed by the depletion.

It might be imagined that the abstraction of so small a quantity of blood as that above prescribed, would produce no effect whatever on so formidable a disease as hypertrophy. Yet experience proves the reverse: it produces a great effect, and I imagine this to be referable, not only to diminished tension of the vascular system, but also to a slight reduction of the rich, stimulant quality of the blood; since I have not found it easy to produce an equivalent impression by mere purgatives and hydragogues, though employed so actively as to produce incomparably more annoyance to the patient.

The diet, in plethoric persons who rapidly reproduce rich blood, should, for the first month or two, consist exclusively of white fish, farinaceous articles, and vegetables: subsequently, a moderate proportion of animal food may be allowed on alternate days. In ordinary, average constitutions, the latter diet may be permitted from the first. In weakly constitutions, and in advanced stages of the disease, when anæmia has either already appeared, or would easily be induced by an insufficiently nutritious diet, animal food should be permitted daily. Whatever be the constitution, the patient should never overload his stomach with an immoderate meal, nor eat heartily during a state of exhaustion from fatigue or fasting, as a degree of palpitation is almost sure to be the consequence. His meals should be evenly distributed, and each should be light. Though three meals, at intervals of five hours, are generally sufficient, a fourth in a light form is better than immoderate indulgence at any one. The food should be perfectly plain and simple; since dyspepsia, by exciting palpitation, greatly aggravates diseases of the heart.

The drink should consist of water, soda-water, or seltzer-water. All stimulants, as spirituous, vinous, and fermented liquors, should be shunned; the only exceptions being, when dyspeptic debility of the stomach demands the addition of a glass of sherry or half an ounce of brandy to a tumbler of water, or when an inveterate habit of free living renders it dangerous suddenly and totally to abstract stimulants: in which case, the patient may gradually be reduced to the least requisite quantity,—which can often be brought so low as one or two glasses of wine in the day. The total quantity of liquids taken should be small, as a considerable quantity bloats the vessels. This remark is peculiarly applicable to the phlethoric. I have frequently found their progress unsatisfactory till they were put upon a *dry* diet.

Any exercise taken, should be so gentle as never to hurry, and, if possible, not even to accelerate the circulation beyond a few beats. Walking up hill is therefore out of question, even though the patient declare that he can do it without inconvenience: riding on horseback is equally objectionable, and staircases should be avoided to the utmost. The pace on level ground should not exceed 2½ or 3 miles an hour for males, and the distance should not be such as to produce lassitude. Gestation in a carriage is unobjectionable.

Purgatives should be used for a week or two with each of the first bleedings, to co-operate with them in making a primary impression. Also, when the action of the heart appears to increase, and yet bleeding is not expedient, three or four copious and watery alvine evacuations should be procured daily by saline aperients, of which none answers better than one or two drachms of sulphate of magnesia in infusion of roses twice or thrice a day. This may be continued for a week or ten days according to the effect; and, in plethoric patients, either the same, or some analogous aperient, should be employed habitually in sufficient doses to keep the body gently open, and to procure, if possible, softish evacuations. When salines are used habitually, their debilitating effects on the intestinal canal may be in a great measure counteracted by adding to the infusion of roses an equal quantity of Comp. Infus. of orange-peel and six or eight minims of dilute sulphuric acid. In patients who are not plethoric, the habitual use of aperients is unnecessary, beyond what may be requisite to procure a single, natural evacuation daily.*

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* A respectable writer, overstraining the principle of draining away the serous part of the blood, has proposed the habitual use of Elaterium as a hydragogue. This,

In addition to purgatives, I have seen the most decided advantage result, in severe cases, from diuretics, and not only when there was dropsy, but also when there was none. Their mode of operation appears to be ultimately the same as that of purgatives: namely, by draining off the serous portion of the blood. I have found many patients, conscious of the benefit which they derived from this class of remedies, in the constant habit of taking cream of tartar, broom-tea, and other similar, popular medicines. One patient, affected with contraction of the mitral valve to the size of an ordinary pea, by these means warded off dropsy, beyond the slightest œdema of the feet, for ten years.

When decided dropsy appears, it must be combated by the most efficient diuretics—the supertartrate, acetate, hydriodate, and nitrate of potass, squill, juniper, digitalis, spirit of nitric æther, tincture of cantharides, decoction of broom, &c., with mercury if not contra-indicated. As no class of remedies is more variable and uncertain than this, when one fails another should be resorted to; and it not unfrequently happens that a weaker is more successful than a stronger. Should diuretics wholly fail, hydragogue purgatives, as elaterium, tincture of jalap, infusion of senna with tartrate of potass, &c. are often invaluable substitutes.

Acetate of lead, in full doses, possesses a powerful sedative action on the vascular system; but, as it is a remedy which, if long administered, is apt to derange the alimentary canal, its employment is not desirable in so protracted a disease as hypertrophy. Fortunately, it can be dispensed with.

Many patients have consulted me after having undergone a course of hydriodate of potass, given with the view of creating absorption of the heart, as it does of glandular structures; but I never could ascertain that the least benefit had been derived from its use.

The state of the stomach and of the biliary secretion should never be overlooked in hypertrophy, as their derangements are amongst the most efficient exciting causes of palpitation. The remedies suitable for dyspepsia and derangement of the liver are

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however, would not only be intolerable to the patient for any considerable period, but would be apt to irritate the mucous membrane. Finally, according to my observation, it is unnecessary—except in cases of obstinate general dropsy, as will presently be explained.

therefore to be resorted to. I deem it unnecessary here to enlarge on them, and on the treatment of dropsy, cough, dyspnœa &c., as these subjects will be found fully discussed in the chapter on disease of the valves.

It frequently happens that, notwithstanding the most judicious use of the means specified, the irritability of the nervous system frustrates their tendency to reduce and tranquillize the action of the heart. In this case, sedatives are eminently useful, and I now resort to them from the first in all cases where there is a considerable disposition to palpitation. Their occasional use, however, is generally sufficient. I have often found excellent effects result from tincture of digitalis to the extent of m. xx or xxx twice or thrice a day; from a drachm of tinct. of hops in mist. camph. administered as often; from three, four, or more grains of extr. of hyoscyamus or conium once or twice a day, from acetate or muriate of morphia, and from these variously combined. The emplast. belladonnæ is also useful.

It must never be forgotten that the irritability of the nervous system and the palpitation may be referable to anæmia,—indicated by its usual signs, a pallid complexion, quick jerking pulse, debility, &c. In this case, it is in vain to resort to sedatives, except as auxiliaries: the true remedies are, full doses of any of the stronger preparations of iron, as the mist. ferri comp. or the ferri sesquioxyd, &c.; aloetic aperients to regulate the bowels; and under-dressed animal food at breakfast and dinner. When, by these means, the due proportions of fibrine and red globules have been restored to the blood, and the anæmic palpitation and irritability have been reduced, the treatment for the hypertrophy may be prosecuted on the foregoing general principles, except that bleeding will rarely be necessary, and the allowance of animal food may still be liberal. In fact, the art of treating hypertrophy consists in keeping the patient rather low, and the circulation very tranquil, yet short of producing anæmia and debility. So far from debility being induced by the measures recommended, I have generally found patients express themselves as feeling lighter and more active. Under these circumstances of calmness without debility, the heart possesses a surprising power of reverting to its natural size,—a power, which it was long before I trusted myself to believe, and which is still disbelieved by the bulk of those, who have not proved it by personal observation. Yet it is not very incredible when we reflect on the rapidity with which external muscles, (especially the hypertrophous muscles of the legs in dancers, of the arms in smiths, &c.) become emaciated and feeble, when exercise of them is wholly suspended.

The above, and indeed every other, mode of treatment is unavailing, if not *steadily* pursued; and it must be pursued for one, two, or three years according to circumstances. The great majority of recoveries I have found to take place between one and two years, but a year or two of subsequent precaution is most desirable, to prevent a relapse. Two of the greatest impediments to success are, first, that the patient is often so much relieved at the end of two or three months, as to believe himself well: secondly, that other practitioners, finding the heart calm and the respiration free, persuade him that he has not, and never has had, organic disease of the organ. But "*we must be afraid of allowing ourselves to be deceived by the perfect calm which blood-letting and abstinence sometimes very promptly produce*" (Laennec).

Though the treatment is prolonged, it is one which trenches exceedingly little on the convenience and comfort of the patient, and he is in general well contented to compromise, on terms so easy, for emancipation from so formidable a disease.

As hospital patients do not remain long under observation, it is only from private practice that an estimate can be formed of the success of the above treatment. The cases which I have collected from this source during the last ten years, afford me reason to believe that nearly the whole who are under the age of 40, may be radically cured, provided the hypertrophy is exempt from complication with valvular or aortic disease, adhesion of the pericardium, softening of the heart, or other organic obstacles to the circulation; and provided also, that the constitution is sound and the general health tolerably good. The few exceptions that occur, are principally those in whom the hypertrophy is very great, and has advanced to the stage producing dropsy and much deterioration of the general health. In persons under the age of 25, even this degree not unfrequently admits of being cured. All degrees and varieties yield more easily in the youthful; and before the period of puberty, it is not uncommon for a moderate degree to be cured, although bleeding be resorted to only at long intervals, as from six weeks to three months. After the age of 40, the curability of the disease is somewhat less, though it is not till the age of 50 or 55 that the difficulty becomes considerable. At this age, I have found that, though hypertrophy can be diminished, and its urgent symptoms in a great measure removed, yet the patient continues under the necessity of permanently maintaining a quiet, peaceable mode of life; as, otherwise, the disease returns.

The treatment described has the advantage of being suitable not only for pure hypertrophy, but for the disease when complicated with valvular or other impediments to the circulation. For the hypertrophic part of the disease may be diminished, and sometimes removed,—in which case the valvular or other impediment, (assuming that neither dilatation nor softening supervene,) occasions comparatively little inconvenience. As, however, a valvular impediment is irremediable, the cure cannot be radical, and the patient remains permanently under the necessity of adhering to a quiet mode of life.

With respect to hypertrophy resulting from pericarditis and endocarditis, obviation should be the aim of the practitioner. If acute rheumatism be treated on the principles already described, (p. 179,) inflammation of the heart will not, according to my experience, occur in more than one out of about twelve, instead of in every second or third. If it should occur, a prolongation of the treatment for pericarditis and endocarditis, described at p. 184 and 219, will generally succeed in removing, or, rather, obviating the hypertrophy. When this has actually taken place, and all inflammation has subsided, I know no more suitable treatment than that for hypertrophy in general.

M. Bouillaud countenances the strict and rigorous application of the treatment of Albertini and Valsalva "when the hypertrophy is really enormous," and a slighter degree of it in moderate cases! The possible occurrence of anæmic palpitation he wholly overlooks!—an inconsistency which is unaccountable; since, in another part of his work, he writes elaborately on *chlorotic* palpitation. He is very partial to digitalis. "It is," says he, "incontestably the most efficacious and direct of all sedatives—the true opium of the heart." He has employed it with much advantage on the endermic plan: that is, he applies a blister to the præcordial region, and daily covers the surface with the powder of digitalis, in doses graduated from 6 to 15 grains. "We thus," says he, "diminish the number and force of the heart's beats, as if by enchantment." Patients, however, have a strong prejudice against digitalis, and will not willingly submit to it long, or often, if its depressing effects are rendered very sensible.

APPENDIX TO HYPERTROPHY.

"Considerable doubt has been excited recently by the high authority of M. Cruveilhier as to the real existence during life of such a condition as hypertrophy with contraction. This anatomist believes the diminished cavity to be merely the result of a tonic contraction of the muscular wall of the ventricle in death. 'The concentrically hypertrophied hearts of Bertin and Bouillaud appear to me,' he says, 'to be hearts more or less hypertrophied, which death surprised in all their energy of contractility.'* The hearts of all those examined by Cruveilhier, who died by the executioner, presented to his observation to a great degree the double phenomenon of increased thickness of walls and diminished cavity, and he has observed the same with persons who died a violent death.† On one occasion I was particularly struck with a similar condition of the heart of a donkey which had been accidentally transfixed by a large trocar, whereby the death of the animal was caused in a few minutes. The muscular structure of the heart was singularly dense. It had contracted at its apex quite to a sharp point, and on cutting into it, the cavity of the left ventricle appeared almost obliterated, and the muscular wall much increased in thickness. I have many times, too, observed the fact noticed by Cruveilhier, that the cavity may be easily enlarged or restored to its natural dimensions by introducing the finger and dilating it, or still more easily, if the heart have been macerated in water for a short time previously. This fact is further confirmed by Dr. Budd, who supports the views of Cruveilhier in an interesting paper in the last volume of the Medico-Chirurgical Transactions. In one of Dr. Budd's cases the thickness of the parietes of the left ventricle eighteen hours after death varied from an inch to an inch and a half, on a transverse section made at a distance from the apex of one-third of its length, and the cavity was not large enough to hold the second phalanx of the thumb, and was almost filled by the carneæ columnæ. This heart, in its open state, was put to macerate; *no force was applied to extend it.* At the end of some days, on being folded up, it was found to have dilated very considerably, so that the left ventricle could not then be said to be smaller than natural. Dr. Budd argues against the existence of the diminished cavity from the fact that of eight cases collected by him, no one afforded signs, either during life or after death, of any obstacle to the circulation through the heart. There were no irregularity of pulse, no dropsy during life, no dilatation of the right cavities after death, phenomena which, it may be said, must of necessity be present if there be an obstacle to the circulation in the heart. It is impossible, as he states, to conceive that a left ventricle, which could scarcely hold an almond, should offer no obstacle to the circulation through the heart. Yet Laennec has recorded a case in which the parietes of the left ventricle had acquired the thickness of from an inch to an inch and a half, and the cavity seemed capable at most of containing an almond stripped of its shell. Yet the day before the patient's death his pulse was natural, the breathing perfectly free, 'and nothing,' says Laennec, 'led me to suppose that this man had a disease of his heart.'" (See Cycloped. of Anat. and Physic. Abnormal Conditions of the Heart, by Dr. R. B. Todd, p. 12.)

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* Dict. de Méd. et Chir. Prat., art. Hypertrophie.

† Mr. Jackson and Dr. Budd have observed this state of the heart in persons who died of cholera.

CHAPTER II.

DILATATION OF THE HEART.

SECTION I.

ANATOMICAL CHARACTERS WITH CLASSIFICATION AND NOMENCLATURE OF DILATATION.

THE disease commonly termed dilatation of the heart, consists in an amplification of one or more of its cavities.

Although I have seen the muscular substance healthy in every form and degree of this affection, in general it is not so. For, when the dilatation is great, and the parietes are feeble in proportion to the quantity of blood which they have to propel, the muscle is usually more or less flaccid, and even softened, and in some cases of a deeper red, in others paler or more fawn-coloured than natural (Gillan, Anderson, Mrs. —l—n). The deep red dye is attributable to venous engorgement of the muscular substance, resulting from stagnation of the blood within the heart. The paleness is often connected with general muscular paleness. The softening is sometimes so great that the substance readily breaks up under the pressure of the fingers.

Dilatation occurs with three different states of the ventricular parietes as to thickness: namely, the thickened, the natural, and the attenuated states. It accordingly resolves itself into three natural varieties corresponding with these states.

1. *Dilatation with hypertrophy*, in which the cavity is enlarged and the walls thickened.

2. *Simple dilatation,* in which the cavity is enlarged, and the walls of their natural thickness.

3. *Dilatation with attenuation,* in which the cavity is enlarged and the walls attenuated.

The first variety is identical in its nature with that variety of hypertrophy called *hypertrophy with dilatation:* the only difference consists in the relative degrees of the two affections, and this difference is indicated by giving precedence to the term *hypertrophy* in the one, and *dilatation* in the other. Thus, *hypertrophy with dilatation* denotes a predominance of hypertrophy; whereas *dilatation with hypertrophy* bespeaks a predominance of dilatation. The second variety is perfectly identical with *hypertrophy by increased extent, with natural thickness of the walls;* but it is better to employ the term *simple dilatation* when the dilatation is so great, or the patient so enfeebled, that its symptoms predominate over those of hypertrophy.

Two, or all three of the forms of dilatation are sometimes found together, in different parts of the same cavity. It is sufficient to notice the fact, without perplexing the memory with a distinct appellation for cases of this compound nature.

The anatomical characters of *dilatation with hypertrophy, and simple dilatation,* are described in the chapter on hypertrophy, p. 237. To *dilatation with attenuation* we now direct our attention. It seldom affects one ventricle without the other. The attenuation may proceed to such an extent as to reduce the most substantial part of the left ventricle to two lines in thickness, and the apex to a mere membrane. In a case lately under my observation, the prevailing thickness was two lines, (Lambert,) and a portion of the apex consisted solely of the internal and external membranes, strengthened by a deposition of lymph on the outside. Extreme attenuation is more common in the right, than in the left ventricle. In either, the columnæ carneæ appear stretched and spread. The inter-ventricular septum is, proportionably, much less attenuated and softened than the other parts. Dilatation takes place more in the transverse, than in the longitudinal direction of the ventricles, and it accordingly communicates to the heart an unusually spherical form, so that the transverse diameter of the organ is often as great or greater than the longitudinal, and the apex is rounded off in such a manner as frequently to be scarcely distinguishable. This alteration of shape is the best criterion for determining whether a heart is dilated or not, when the enlargement is so inconsiderable as to render the question doubtful.

When both the auricle and ventricle are much dilated, it is not unusual to find the intermediate aperture widened, and its valve sometimes not large enough to close it. As this causes regurgitation, it is as serious a malady as disease of the valve itself, producing the same effect. It should be understood by the practitioner, because it is apt to be overlooked, *post mortem*, in cases which had presented signs of regurgitation; and, thus, his confidence in valvular diagnosis is apt to be shaken.*

Laennec, although he had never seen a case of rupture of the heart from dilatation, believes, with Burns, that it may occur; particularly, as dilatation is generally attended with softening. I witnessed a case of this kind a few years ago. The patient, who was aged upwards of seventy, fell back suddenly while on the night chair, and immediately expired. A fissure an inch in length was found in the left ventricle, its substance was softened and of a deep violet colour, and the cavity of the pericardium was gorged with blood. Dr. Williams communicated to me the case of a relation of his, who died from rupture of the heart in a somewhat similar way; but the orifice through which the blood escaped was small, round, and encircled by dark ecchymosis. The patient (a lady of fifty-eight) had been subject to severe angina for some months before her death. He suspects that there were both attenuation and softening in this instance, but is not sure, as he did not see the body.

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* The following are M. Bouillaud's measurements of the orifices in their natural and dilated states.

	Mitral.	*Tricuspid.*
Medium	3 inches 6 lines (dilated 4. 1⅓)	3 10 (dilated 5 2)
Maximum	3 — 10	4 0 (— 5 9)
Minimum	3 — 3	8 9 (— 4 2)
	Aortic.	*Pulmonic.*
Med.	2 inches 5¼ lines (dilated)	2 7¾ (dilated 3 2⅜)
Max.	2 — 8 — (— 3 5)	2 10
Min.	2 — 4	2 6

The natural measurements are founded on three or four cases only, which is too small a number to justify implicit confidence in them.

In order to judge accurately of dilatation of the auricles, it is necessary to have distinct ideas respecting their natural form and dimensions. The four cavities of the heart are very nearly equal in capacity; but, as the parietes of the auricles are very thin, and those of the ventricles are thick, the auricles, when simply full and not distended, form only about one-third of the total volume of the organ; or, what is the same thing, the volume of the auricles equals about half that of the ventricles (Laennec de l'Auscult. tom. ii. p. 523). The right auricle, being generally found in a state of distention, and being of a more elongated, flattened form than the left, has the appearance of being considerably larger, though in reality it is only a little so.

Distention, taking place during the last moments of life, and observable, though more rarely, in the left auricle as well as in the right, constitutes the great source of fallacy in determining after death whether these cavities are really dilated or not; for the engorgement, though only of a few hours' duration, may stretch them to a magnitude almost equalling that of the ventricles.

M. Laennec has given good criteria, by which a dilated may be distinguished from a distended auricle. An auricle simply distended is tense, and through its thinnest parts distinctly shows the dark blood within. One dilated, does not present the same appearance of tension, and its parietes are more opake. When the blood is evacuated through the vessels without cutting into the cavities, the latter, if merely distended, return at once to nearly their natural size: whereas, if dilated, they maintain almost the same size which they had when full. Dilatation of the auricles, as already stated, scarcely ever exists without more or less thickening of their parietes.

The method of distinguishing distention from dilatation is much the same in the ventricles as in the auricles: namely, when merely distended, they are found enlarged, firm and tense; but these conditions almost entirely disappear, when the blood is pressed out through the natural apertures. On the contrary, when truly dilated, they have no appearance of tension, are more or less flaccid, and the enlargement persists after the blood has been evacuated.

SECTION II.

MODE OF FORMATION, WITH THE PREDISPOSING AND EXCITING CAUSES OF DILATATION.

Dilatation of the heart is a purely mechanical effect of over-distention. Blood, accumulated within its cavities, exerts a pressure from the centre towards the circumference, in every direction; and when once it surmounts the resistance offered by the contractile and elastic power of the parietes, these necessarily yield and undergo dilatation. The rapidity with which this process takes place, and the extent to which it is carried, depend on the degree in which the distending, exceeds the resisting force: and as the latter bears a direct ratio to the volume of the muscle, supposing it to be healthy, it follows that individuals with *naturally* thin-walled hearts are more prone to dilatation than others: supposing the muscle *not* to be healthy,—supposing it to be flabby or softened, (from general emaciation, anæmia, typhus, scurvy, purpura, inflammation of the heart, or any other cause of softening specified in the chapter on that subject,) its resisting power is diminished by these circumstances, and it is more susceptible of dilatation by the distending power. In any case, those cavities of the heart which have the thinnest parietes, are, cæteris paribus, the most susceptible of the disease. Accordingly we find that the right ventricle is more frequently and promptly dilated than the left, and the auricles than either, when exposed to distending causes.

In order to produce permanent dilatation, the operation of the exciting cause must either be prolonged for a certain time, or frequently repeated at brief intervals. Contraction of an orifice, for instance, acts in the former manner; and nervous palpitations, and occupations requiring constantly renewed and long sustained muscular efforts, produce their effect in the latter way. When the operation of the cause is only brief and transitory, the result is merely a temporary *distention*, from which the muscle recovers itself by its own elastic and contractile reaction so soon as the distending force is removed. This cannot be regarded as a pathological state, and it must, therefore, be carefully distinguished from genuine dilatation.

The causes of dilatation, are, 1st, deficient power of the heart, whether congenital or acquired, in proportion to the system: 2nd, in general terms, all obstructions to the circulation, whether situated in the orifices of the heart, or in the aortic, or pulmonary system. The second class of causes is, in fact, essentially the same as the exciting causes of hypertrophy, independent of inflammation (see p. 245). For, as stated under hypertrophy, it depends on the proportion which the resistance of the muscle bears to the distending force, whether the one affection or the other be produced. When, therefore, dilatation occurs in one of the cavities with naturally thick walls, in which we should more properly expect hypertrophy, it must be ascribed, either to a congenital disproportion of the heart, in consequence of which the cavity in question is thinner, and therefore more disposed to dilatation, than natural; or it must be attributed to the obstruction, from its nature or situation, bearing more in proportion on that particular cavity, than on any other. It is from overlooking these considerations, respecting the relations of the resisting and distending forces to each other, that some have excluded dilatation from the catalogue of mechanical diseases, and supposed that it takes its rise in any cavity of the heart either by chance, or by some vital predilection, some vague, unintelligible predisposition.

Dilatation occasionally affects only a single ventricle, and it is generally the right, and seldom in a great degree: much more commonly it attacks both, and then the degree may be greater in either. The auricles, being protected by their valves from the direct influence of the numerous causes of pressure which operate on the ventricles, are far more exempt both from dilatation and hypertrophy. But when the auricular valves are diseased, whether their state be that of contraction, which impedes the transmission of the auricular blood, or of permanent patescence, which allows a regurgitation of the ventricular, the auricles, suffering unnatural distention, become dilated.

It is seldom that dilatation of the auricles occurs under any other circumstances than those of disease of their valves: so seldom, indeed, that Laennec does not recollect to have seen an instance, though he does not deny the possibility of the occurrence. More instances than one, however, have fallen under my own observation, and I have generally found the dilatation connected with some circumstances which rendered the ventricle incapable of freely evacuating its contents. It is natural, indeed, to suppose, that, when such is the case, the stagnation of blood in the ventricles must, for the time, have an effect in distending the auricle equivalent to that produced by contraction of the auriculo-ventricular valve; and, considering the frequency of stagnation in the right ventricle, we might at first expect dilatation of the corresponding auricle from this cause, to be frequent. But it must be remembered that, for the production of the disease, it is necessary that the operation of the cause be permanent, or at least very prolonged. Such, however, is seldom the case with the stagnation in question; for a ventricle, though so feeble in itself, or so encumbered by an obstacle before it in the course of the circulation, as to become gorged during an accelerated state of the heart's action, will often, when tranquillity is restored, transmit its contents with a facility that could scarcely be anticipated. During such intervals, therefore, the muscular fibres of the auricle recover their contractile power, and restore the cavity to its natural size. Hence it is, that, though dilatation of the auricles is occasionally a result of ventricular engorgement, it is much more commonly dependent on imperfections of the auricular valves.

SECTION III.

PATHOLOGICAL EFFECTS OF DILATATION, AND MODE OF THEIR PRODUCTION.

"Authors," say M. M. Bertin and Bouillaud, "have entered into long disquisitions on what are called the general symptoms of dilatation or aneurism of the heart, and they have allowed themselves to fall into great errors in considering dilatation a *primitive* malady, instead of regarding it as consecutive to another lesion, which was the source of the symptoms that they attributed to the dilatation itself. We have already proved, continue they, that this malady *necessarily supposes an obstacle to the course of* *the blood;* but this obstacle, at the same time that it gives rise to an aneurism of the heart, produces other striking phenomena, such as engorgement of the vessels, serous infiltration, passive hæmorrhages, &c. These phenomena have been taken for the effects of the dilatation of the heart, while this has, strictly speaking, no other relation with them than as being a result of the same cause, that is to say, of an embarrassed state of the circulation" (p. 382).

I cannot concur with M. M. Bertin and Bouillaud in these opinions. It is true that, in order to produce dilatation, there must exist a weight or pressure of the circulation upon the heart greater than the organ is capable of sustaining: and it is true that such pressure may be occasioned by the mechanical obstacles to which M. M. Bertin and Bouillaud ascribe it: namely, contraction of the orifices of the heart, diseases of the aorta, and all maladies which impede the course of the blood, whether in the lungs, or in the system of the great circulation (p. 380). But it is equally true that the same pressure on the heart may result, not from increased weight of the circulation, but from deficient power of the heart; and such is its cause in those, who, by original conformation, have the organ thin, in proportion to the size of the body. I believe that this is a more powerful and certain cause of dilatation than the impediments alluded to by Bertin and Bouillaud; for the malady prevails principally in the female sex, in whom the walls of the heart are, in general, thinner than in men: though women are less exposed than men to the exciting causes of dilatation, as they lead a more tranquil, temperate life, and are less subject to diseases of the arteries and valves. Another class in whom debility of the heart exists as a cause of dilatation, comprises those who have had the organ softened or otherwise enfeebled by disease: an effect not unfrequently produced by typhoid fever, by inflammation of the substance of the heart, and by other causes specified in the chapter on Softening.

Dilatation, then, occurring under the circumstances described, is as justly entitled to the rank of a *primitive* disease, as hypertrophy: for as, in both, the disease depends, not on the pressure of the circulation, but on the manner in which the heart resists that pressure; in both, the organ itself is the part where the disease originates: the only difference being, that the effect is produced in the one case by deficient, and in the other, by superabundant power of the muscle.

In the next place, M. M. Bertin and Bouillaud have, in my opinion, attributed far too much to the mechanical obstacle of which they consider dilatation to be the effect, when they say that this obstacle is the sole cause of all the symptoms, which authors have been in the habit of ascribing to dilatation; namely, passive congestions and hæmorrages, dropsy, &c. It is true that when the obstacle is so great as to constitute an extreme impediment to the circulation, it may produce the symptoms in question; but it does not produce them, or only in a very slight degree, when the impediment is not extreme. I have repeatedly witnessed cases in which a well marked, if not a considerable obstacle, as a contracted valve, a regurgitation, or a dilatation or aneurism of the aorta, had subsisted for a long period, even for years, without producing any material symptoms of an obstructed circulation; but the moment that dilatation of the heart supervened, the symptoms made their appearance in an aggravated form. I apprehend, therefore, that the heart is the part mainly concerned in their production: nor do I think this opinion less tenable because the symptoms are more severe when dilatation of the heart co-exists with an obstacle, than when the dilatation exists alone; for it is natural to suppose that, when two causes conspire to produce the same effect, that effect should be greater. But this is not all; for not only does each produce its own effect, but one increases the effect of the other: namely, the obstacle adds so much to the pressure of the circulation on the heart, that this organ labours under a double disadvantage, first, from its own diminished power, and secondly, from a preternatural pressure upon it. Thus the resulting effect of the obstacle and the dilatation of the heart combined, is greater than the sum of the two taken separately.

In further invalidation of M. M. Bertin and Bouillaud's opinion, I may add that I have not only seen numerous cases of a mechanical obstacle unattended with passive congestions, dropsy, &c.; but I have seen a still greater number of instances in which all the phenomena of an obstructed circulation were occasioned by dilatation alone; as no other obstacle capable of accounting for them, could be detected in the course of the circulation. Such cases, in fact, are of ordinary occurrence, and, when flabbiness or softening co-exists with dilatation, the effect is still more marked.

According to the foregoing arguments, then, it appears, 1. that dilatation may be a *primitive* disease; and that, as such, it is capable of producing all the phenomena of an obstructed circulation. 2. That when it is consecutive to another lesion, it plays a prominent, and perhaps even, in some cases, a more important part than that lesion, in producing the phenomena of an obstructed circulation.*

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* M. Bouillaud, in his Treatise in 1835, avows himself to be the author of the opinions which I controvert. In reference to them, and similar ones applying to hypertrophy, I stated in the Introduction to this work, (p. xiv,) that M.M. Bertin and Bouillaud considered "*the symptoms of a retarded circulation to be, under* ALL *circumstances, the result of a mechanical obstacle to the course of the blood.*" M. Bouillaud complains, in his Treatise in 1835, (vol. i. p. 267, note,) that I have, in these words, mis-stated his opinions, and he cites, in proof, the following passage from his conjoint work with Bertin, in 1824. "It is very clear that, *considered in an abstract manner*, (these words in italics M. Bouillaud omits in his citation) dilatation of the heart has the effect of enfeebling the contractile power of the muscular substance of this organ, by reason of the distention which it causes the organ to undergo. The muscular fibres lose, as it were, in force, what they gain in length. Thus, then, if we conceive of this dilatation, *abstracting* (*en faisant abstraction*) *the cause which has occasioned it*, we *should* give (donnerions) to it, as signs, feebleness and softness of the pulse, dropsies and passive hæmorrhages, in a word, all the phenomena which we know to be the result of an obstacle to the circulation" (Traité de Bertin et Bouillaud, p. 384). "I am, then," says M. Bouillaud, in his remarks appended to this citation, "agreed with Dr. Hope on this point—that dilatation can, *of itself*, produce the symptoms of obstruction or embarrassment of the circulation."

I rejoice to find that M. Bouillaud makes this admission, because I sincerely believe that he has done an injury to practice by maintaining the opposite opinion. But, as he has charged me with misrepresenting his opinions, I am now under the necessity of showing that he has arrived at his admission at the expense of contradicting himself in every other part of his works, both in 1824 and 1835. In doing this, I shall simultaneously show that I have given a fair statement of his opinions.

He has not accurately quoted the above passage from the work in 1824. He has suppressed the clause in italics at the commencement of the passage, has stopped at a semicolon, instead of concluding the sentence, and has not alluded to the previous sentences. The introduction of these omissions reverse the meaning of the whole passage. The reader shall judge for himself. In the previous sentences he says, "I have shown above that dilatation of the heart was *the mechanical result of a cause* which occasioned engorgement of the cavities of the organ, and *that it was to the action of this cause*, instead of to the dilatation itself, that we were to refer divers symptoms which authors have attributed to the latter. The dilatation being pro-

As, in cases of dilatation combined with a mechanical obstacle, it is impossible "to assign to the dilatation and the obstacle, the exact proportion which each bears in the production of the same

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duced, we must now examine what may be its influence on the system of the circulation. Now, it is very clear that, (here comes the suppressed clause,) *considered in the abstract*, dilatation has the effect of enfeebling, &c." The sentence then concludes thus:—"; but as the *cause* of the dilatation is itself capable of producing all these phenomena, it is very difficult to assign, to the dilatation and to its cause, the exact part which it takes in the production of the same effects." The import of the passage, now, stands thus:—Dilatation has always a mechanical cause: if, by a stretch of imagination, we could conceive of the dilatation as abstracted from its cause, we should assign to it certain phenomena; but, as this abstraction is wholly a flight of imagination, we must assign the phenomena to the *cause* of the dilatation itself, admitting, however, that the dilatation, when once produced, may co-operate in the generation of the effects. The same ideas are expressed, in the most unqualified terms, in another passage from the same Treatise, (viz. of 1824,) quoted above at p. 299; and again, in a third passage, at p. 227 of the Treatise: namely, "many authors have stated these signs, (viz. of an obstructed circulation,) as being peculiar to aneurism of the heart: *it is a great error*; for, far from the aneurism of the heart being the *first* mover of the phenomena which we observe, *it is itself only one of the effects*, and, so to speak, one of the *accidents*, of contraction of the orifices"!

Similar statements run throughout M. Bouillaud's work in 1835. For instance, in vol. ii. p. 216, note, he repeats the passage just quoted. The same, in rather different words, is reiterated at p. 613. The same, in reference to *active* aneurism, (hypertrophy,) is repeated, in *unqualified terms*, at p. 445. Viz. "Passive congestions, whether of blood or serum, do not in reality occur, except in cases where hypertrophy is complicated with other lesions capable of opposing an obstacle to the course of the venous blood,—as contractions of the orifices or cavities of the heart, and important organic lesions of the principal arteries or veins." He reiterates the same, in equally unqualified terms, at p. 447, and represents himself to have exposed a general error of authors! The only resemblance to a qualification that I can find in the whole work, is in the chapter on Dilatation, p. 532, where, (as if he had recently been reading my remarks in the text above,) instead of saying, *in* ALL *cases*, he only says, "*in the* IMMENSE MAJORITY *of cases, the above accidents* (i. e. *passive congestions, dropsy, &c.*) *are referable to a mechanical obstacle to the course* of the blood, which is at the same time the cause of the dilatation." Yet even this qualification he annihilates in the next sentence: for he adds, "not that dilatation does not *sometimes* play a *certain* part (never, therefore, the whole) in the production of the accidents; . . . . . but, once more, its influence has been exaggerated; and, in a number of cases, hypertrophy does more than compensate for the weakness which might result from dilatation." If any doubt remain as to M. Bouillaud's opinion, the following passage is a coup de grace. "We see here again that M. Louis uses the word *aneurism* in the vague acceptation which it has received from certain authors. *No, a hundred times, no!* Most of the symptoms to which M. Louis here alludes, are not, correctly speaking, those of *aneurisms* or dilatations of the heart, but, in fact, *those of a lesion of the valves with obstacle to the circulation.* If I expose this error *again and again*, it is because it still reigns generally in the minds of observers otherwise the most distineffects,"[1] it is necessary, in order to ascertain the real effects of dilatation, to confine ourselves, in studying them, to the simple, uncomplicated form of the disease.

Taking into consideration this form alone, and admitting, on the foregoing grounds, that it is capable of producing all the phenomena of an obstructed circulation, we have next to inquire how or by what mechanism it produces them. To answer this question,—it produces them by putting the muscular fibres of the heart preternaturally on the stretch, whereby their contractile power is diminished: "they lose, as it were, in force what they gain in length;" and it is this deficiency of power in the main spring of the circulation which constitutes the obstacle, if it may be so called, to the circulation; in the same way that weakness of the spring of a time-piece retards its movements.

It must be distinctly understood that these observations do not apply to dilatation with which a predominant degree of hypertrophy is conjoined, for the heart then acquires more force in virtue of the hypertrophy, than it loses by the dilatation, and the consequence is, an increased, instead of a diminished energy of the circulation. Less hypertrophy than is generally supposed, suffices to occasion this increased energy. It is not even essential that the walls of the heart be thickened at all, provided the muscular fibre is healthy, the constitutional powers unimpaired, and the dilatation moderate, that is, not so excessive as to be greatly out of proportion to the thickness of the walls. It is in consequence of such cases being attended with increased energy of the circulation, that it has been necessary to transfer them from the class of dilatation to that of hypertrophy, where they

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guished, and because one cannot make truth triumph except by defending it with perseverance" (Traité, ii. p. 573, note)!

Notwithstanding all the above, M. Bouillaud comes, with unaccountable inconsistency, to the conclusion, "I am agreed, then, on this point with Dr. Hope . . . . namely, that dilatation can, *of itself*, produce the symptoms of obstruction or embarrassment of the circulation" (vol. ii. p. 268, note)! If, however, M. Bouillaud does me the honour of agreeing with me, he must correct all the passages which I have quoted. Till then, with the utmost anxiety to be just, I cannot comprehend that I have mis-stated his opinions; and I should not have dwelt on this subject, had it not been from anxiety to check a most important error, which might easily become current under the shade of so justly authoritative a name as that of M. Bouillaud. That portion of the error which applies to hypertrophy, has already been refuted at p. 253.

constitute the variety called ***hypertrophy by increased extent, without thickening, of the walls.***

M. M. Bertin and Bouillaud conceive a case in which, the heart gaining, in virtue of its hypertrophy, precisely as much as it loses by reason of its dilatation, there results a sort of compensation or equilibrium, which maintains the functions in their healthy condition (p. 385). It would be erroneous, however, to suppose that this is not a state of positive disease; for, though the functions may be adequately performed while the circulation is tranquil, whenever it is hurried, the heart, either, unable to contend with the increased pressure of the blood, becomes gorged; or, struggling against, and surmounting the obstacle, it palpitates violently, contracts beyond its normal degree, and expels an excess of its contents with preternatural force. In either case, the lungs become congested,—in the former, from retardation of the blood in the pulmonary veins,—in the latter, from an excessive influx through the pulmonary artery; and in either case an attack of dyspnœa is the consequence.

SECTION IV.

SIGNS AND DIAGNOSIS OF DILATATION.

In the preceding section I have shown that the effect of dilatation is, to enfeeble the heart, and thereby occasion the phenomena of an obstructed circulation. We have now to examine those phenomena as signs of dilatation.

General Signs.—The heart, when weakened by dilatation, is subject to palpitations of a feeble, oppressed kind, and more or less distressing, frequent, and prolonged, according to the extent of the malady. In general, they are protracted. The attacks are provoked by any over-exertion or mental excitement.

The pulse is soft and feeble, and, if the debility of the heart be very considerable, it is small. Irregularity and intermittence are rare, except during protracted and distressing paroxysms of dyspnœa, or when the vital powers are much exhausted, as in the advanced stage of the disease. When, however, softening accompanies the dilatation, I have found that the pulse is apt to be as small, weak, intermittent, irregular and unequal, as in the worst cases of disease of the mitral valve, with which, for this reason, softening is frequently confounded. (See Softening, for the Diagnosis.)

The languor of the arterial circulation in dilatation causes the extremities and surface to be chilly, the disposition to be melancholy, and the character to be deficient in energy.

The blood, not being freely transmitted by the left ventricle, accumulates in the lungs by retardation: whence difficulty of respiration; cough, sooner or later attended, in many cases, with copious expectoration of thin, serous mucus; œdema of the cellular tissue of the lungs, greatly aggravating the dyspnœa; terrific dreams with starting from sleep; and passive, pulmonary hæmorrhage of dark, grumous blood in small quantities, forming sanious sputa, and generally the precursor of death in individuals affected with great difficulty of respiration. After death, I have often found this hæmorrhage connected with pulmonary apoplexy, and always with great engorgement.

The lungs being obstructed, the engorgement is propagated backwards to the right side of the heart, to the great veins, and finally to all their ramifications. From this venous engorgement arises a series of striking phenomena, which we shall review successively, premising that the hæmorrhages and dropsy do not generally come on till a late stage of the disease.

1. *Serous infiltration.* This generally makes its appearance first in the lower extremities, because it is in them that the circulation is most languid, the return of the blood being opposed by its gravity, while it is little promoted by the action of superincumbent muscles. The œdema gradually ascends, and, under the name of anasarca, may eventually attain the utmost degree over the whole surface of the body. Increased serous exhalation takes place from the serous membranes also: whence, hydrothorax, hydro-pericardium, and ascites; one or other of which is almost invariably present when there is much external dropsy.

2. *Discoloration of the face.* If the complexion was originally florid, it becomes purple or deep violet, on the centre of the cheeks, the end of the nose, and the lips, with intumescence of the latter,

while the intermediate parts are pallid and sallow. If originally pale, it becomes cadaverously exsanguine, and has a dusky, leaden or venous cast, especially around the eyes. The lips are either livid, or very pale. Lividity sometimes shows itself in the extremities as well as in the face.

3. *Congestion of the brain.* This produces the usual symptoms of passive cerebral congestion, and of the corresponding form of apoplexy; namely, dull headache, felt principally along the course of the great sinuses; hebetude of the mental faculties; stupor, convulsions, and eventually complete coma. It is not unusual for these symptoms to supervene a few days before the fatal termination. Sometimes they depend, not on congestion alone, but partly also on serous effusion into the ventricles, or on the surface, resulting from the congestion; sometimes, again, the congestion ends in sanguineous apoplexy, of which I have seen several instances. Whence it is incorrect to suppose that this catastrophe is peculiar to hypertrophy of the heart.

4. *Injection of the mucous membranes.* It is common to find them after death so vascular as to present the appearance of inflammation. This is especially the case in the stomach and intestines, and it is necessary to be aware of the circumstance, in order to guard against the error of attributing the redness to inflammation.

5. *Passive hæmorrhage.* This takes place from the lungs, as already stated: also from the nose, the stomach, the intestines, the uterus, and more rarely from the bladder. It results from engorgement of the mucous membranes. The effusion consists of dark blood exuding in small quantities. When from the stomach, and not immediately ejected, it has occasionally the appearance of coffee grounds, in consequence of being exposed to the coagulating action of the gastric juice. In the intestines, it is often blackened by the intestinal acids,—the carbonic, acetic, and sulphuretted hydrogen.

6. *Congestion and enlargement of the liver.* This is so common a consequence of retardation of the blood on the right side of the heart, that few persons so affected in any considerable degree, are exempt from it. This has, I believe, been almost entirely overlooked by authors on the diseases of the heart, and it

is still very little known. By the obstruction which it occasions in the system of the vena porta, it leads to ascites and jaundice; also eminently favours hæmetemesis, intestinal hæmorrhage, piles, and, though indirectly, uterine hæmorrhage,—many cases of which I have found to be obstinate till the hepatic enlargement was reduced by mercury and aperients. This latter fact has been noticed by Dr. Locock.

7. *Angina of the heart* may occur as an adventitious complication of dilatation, no less than of hypertrophy (p. 268).

Such are the general signs of dilatation of the ventricles. I may here mention that I have met with two or three cases in which dilatation of the left ventricle caused mitral regurgitation, with murmur, simply by rendering the orifice too large to admit of its being closed by the valve. The first case which drew my attention to the fact was that of a horse, which Mr. Field, the eminent veterinary surgeon, requested me to see. It presented the usual murmur of mitral regurgitation, and the small, weak, irregular, unequal, and intermittent pulse characteristic of that affection. On post-mortem examination, Mr. Field pronounced the mitral valve to be healthy, but too small to close the orifice, in consequence of the latter being enormously dilated, in connexion with general dilatation of the ventricle.

In another case, a gentleman, he *completely recovered from mitral regurgitation with murmur, and most severe general symptoms.* Whence I infer that the regurgitation resulted from great dilatation, which was a leading feature of his case, and was removed by the treatment presently to be described. In a third case, what seems to be a similar affection, is in progress of cure.

General Signs of Dilatation of the Right Ventricle in particular.

The signs which Corvisart regards as the most certain, are, greater dyspnœa than in affections of the left ventricle, a more marked serous diathesis, more frequent hæmoptysis, and a greater lividity of the face, sometimes reaching a dark violet hue. There is no doubt that these may be effects of dilatation of the right ventricle; but they are not indicative of that affection in particular, because they are produced equally by hypertrophy with dilatation of the same cavity, and by valvular disease on the left side of the heart, especially mitral contraction and regurgitation. As Corvisart was a total stranger to the latter, I have little doubt that, in many of his cases, the symptoms which he ascribed to dilatation of the right ventricle, really belonged to the valvular disease. In any of these affections the colour is not an essential sign; for it depends, as I have repeatedly explained, upon the original complexion; and so far from being always livid or purple, it is very common, in cases of great dilatation of the right ventricle, no less than of the left, to see the face deadly pale, and the lips exsanguine.

The sign which, with Laennec, I think the most constant and characteristic of the *equivocal* signs of dilatation of the right cavities, is, permanent turgescence of the external jugular veins, *without sensible pulsation.* This turgescence does not disappear when the vein is compressed at the upper part of the neck, and the influx of blood thus prevented.

Although all these signs of dilatation of the right ventricle are equivocal of themselves, they have some weight when coinciding with the evidence of auscultation; and by the two classes of signs combined, dilatation of the right ventricle, when considerable, may often be detected with tolerable success. I must admit, however, that the differential diagnosis is of little importance, provided we can detect that there is dilatation somewhere, which is generally very possible. Whether the dilatation be connected with valvular contraction on the left side of the heart, is an ulterior question, to be determined by ascertaining whether there exist the characteristic signs of that contraction. (Vid. the chapter on disease of the valves.)

General Signs of Dilatation of the Auricles.

This affection presents no general signs distinguishable from those of the disease in the corresponding ventricle or valve to which it owes its origin; but its existence may safely be inferred when the valve in question is either much obstructed, or permanently open; or when, from any cause, there is great retardation of blood in the ventricle.

Physical Signs.—The rationale of the impulse and sounds of

Dilatation are explained at p. 68. The signs of the two first varieties of dilatation; namely, *Dilatation with Hypertrophy*, and *Simple Dilatation*, that is, with a natural thickness of the walls, are given in the chapter on Hypertrophy, under the head of *Hypertrophy with a predominance of Dilatation* (see p. 273 for the impulse, and p. 276 for the sounds). It only remains for me to describe the signs of the third variety, or *Dilatation with Attenuation.*

The Impulse.—In this variety the impulse is diminished, and in extreme cases entirely absent, even during palpitation. When felt, it is only a brief percussion of the thoracic parietes, not elevating the ear. When the dilatation is great, the impulse is a little lower down than natural. It sometimes happens that, of several beats of the heart that are *heard*, one only is *felt*, and if this is vigorous, it warrants a conclusion that the parietes are little attenuated. Though Laennec does not make this observation, I have assured myself of its accuracy by numerous post-mortem examinations. When the impulse in any form of dilatation is felt over the lower part of the sternum, it denotes dilatation of the right ventricle, but not with certainty.

The Sounds.—When the walls of the ventricles are merely thin without being dilated, the first sound is louder, shorter, and clearer than natural: it approximates in its character to the second sound,—that produced by the extension of the semilunar valves, and which is analogous to the flapping of a pair of bellows, or a gentle tap on the hand with a finger. When there is dilatation with attenuation, even in a moderate degree, the first sound becomes almost the same, and nearly as strong, as the second; and, finally, when the dilatation is considerable, the two sounds cannot be distinguished either by their nature or intensity, but solely by their respective situations, (the first over the lower half of the ventricles, and the second over the semilunar valves, opposite to the lower edge of the third rib, and thence up the great arteries,) and by their respective relations of synchronism or anachronism with the arterial pulse : and, as the pulse in remote arteries, as the radial, is, in dilatation and other diseases of the heart retarding the circulation, later than the ventricular systole and first sound, in a degree greater than natural, the pulse of the carotid or subclavian should be felt. The second sound in dilatation is increased, except when the heart is enfeebled by dilatation with attenuation or softening.

In proportion as the sounds of the heart are louder, they are audible, cæteris paribus, at a greater distance over the chest: accordingly, M. Laennec has proposed a scale by which the extent is made an index of the degree of dilatation and attenuation. Before describing this scale and showing its fallaciousness, it is necessary to acquaint the reader with the range of the sounds in the natural state.

In a healthy man, of medium stoutness, and whose heart is in the best proportions, the sounds, according to Laennec, are audible in the præcordial region alone; that is, in the space comprised between the cartilages of the 4th and 7th left ribs, and underneath the inferior half of the sternum; also, if the sternum be short, in the epigastrium. In the first edition of this work I expressed my opinion that they might be heard beyond this range. I have subsequently assured myself that there are very few cases in which the second sound cannot be traced along the course of the ascending aorta and pulmonary, and heard above the corresponding clavicle on either side,—a fact very intelligible since it has been demonstrated by my experiments, (p. 25,) that the second sound is occasioned by the semilunar valves. That its transmission to the clavicles takes place principally through the medium of the aorta and pulmonary, is countenanced by a case, brought to me by Dr. Blundell, in which an aneurism of the ascending aorta, pulsating between the second and third *right* ribs, had pulled the aorta, and with it the pulmonary artery, very much to the right side; whence the second sound could not be traced along the natural course of the pulmonary artery, nor scarcely heard above the *left* clavicle; while, above the right, it was perfectly distinct. There can be no doubt, however, that the transmission is assisted by the sternum and other solids.

The sounds, explored on the heart itself, are, according to Laennec, "similar and equal on the two sides," those of the right being most audible under the sternum, and those of the left, under the cartilages of the ribs. I have myself, however, strong reasons to believe that the first sound of the right ventricle is shorter and smarter, (*i.e.* more flapping,) than that of the left;

because the walls of the right ventricle are thinner, and their state, therefore, more analogous to that of dilatation (See Conclusions on the sounds, p. 46).

When the sounds are audible beyond the limits mentioned by Laennec, they are heard successively in the following places, constituting his scale alluded to: viz.

1st. Along the sternum and at the left superior anterior part of the chest as high as the clavicle;

2d. Over the same extent on the right side;

3d. The left side of the chest, from the axilla to the region of the stomach;

4th. The right side over the same extent;

5th. The posterior left side of the chest;

6th. The posterior right side.

The intensity of the sound is progressively less, according to Laennec, in the succession indicated, provided the parts around the heart are in the same relative states. But there are so many diversities in these, which may interfere with the order described, that I have found the scale of M. Laennec of little practical utility in estimating the degree of dilatation. Thus, in very fat subjects in whom the impulse of the heart is not perceptible to the hand, the space over which its sounds can be heard by the cylinder, is much more limited than natural: Laennec has even found them confined, in some instances, to about a square inch, though I cannot say that this has occurred to myself.* On the other hand, "in meagre persons," says Laennec, "in those who are narrow-chested, and in children," the sounds are audible much further: namely, "over the two inferior thirds, or even three-fourths of the sternum, sometimes even over the whole of that bone and at the left anterior superior part of the chest as high as the clavicle; often, also, though less distinctly, below the right clavicle." In very meagre subjects I have heard them over the whole chest, both posteriorly and anteriorly.† I have also frequently heard the first sound below the umbilicus, when exploring pregnancy. Now, as it is almost impossible to make an exact estimate of the degree in which stoutness limits, and leanness, &c. extend the range of the sounds, this range is not a sure criterion of the degree of dilatation.

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* M. Bouillaud has more recently expressed a similar opinion. "In very fat subjects, the sounds of the heart are less extensively audible than in the meagre; but I dare affirm that M. Laennec has deviated a little from the truth in saying"—"that the space over which they may be heard with the aid of the cylinder, is sometimes confined to a surface of about a square inch" (Bouillaud, du Cœur, i. 107).

† This observation also has been corroborated by M. Bouillaud in his recent work:

Again, a lung in any way consolidated, whether by hepatization, tubercles, or compression by fluid in the cavity of the pleura, transmits the sounds of the heart more strongly than a lung that is sound and permeable to air—a phenomenon explicable on the principle that dense bodies are the best conductors of sound. The effect is the same though there be cavities in a tuberculous lung; for the sound is transmitted, not through the cavities, but through their walls, which are denser than healthy pulmonary substance.

Under these various circumstances, then, the sounds are irregularly propagated, and the progressive scale of Laennec is interfered with. For instance, if the right lung be consolidated, the sounds will be more audible on that side than on the left.

My own mode of estimating the degree of dilatation, is, by observing how far the first sound resembles the second, and comparing the intensity of the first, heard immediately over the ventricle affected, with what I conceive, from experience, would be its intensity in the same subject if the heart were healthy. I used formerly to corroborate the estimate, if necessary, by the scale of Laennec; making allowance, as far as is practicable, for stoutness, leanness, youth, pulmonary condensation, &c.; but the experience of the last seven years having more strongly convinced me of the practical inutility of that plan, I now seldom resort to it.

The manner in which I judge of attenuation by the first sound, is less by its loudness, than by its greater shortness and clearness —its more complete assimilation to the second sound; for I think

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"I can certify," says he, "that in the subjects belonging to the category specified by Laennec, I have, not once only, but many hundred times, heard the sounds of the heart, not only in the regions indicated by Laennec, but in all other parts of the chest, without excepting even the right posterior region: also, at the lateral parts of the neck, a situation where they are very often almost as loud as in the præcordial region itself." "The transmission of the sounds," he continues, "takes place through the walls of the chest and the contained organs: also, through the vertebral column, which is articulated with the ribs" (Traité, i. 107).

it is often louder in dilatation with hypertrophy, or even with a natural thickness of the parietes, than with attenuation. This opinion is opposed to that of Laennec, who " thinks he may regard it as constant, that the extent over which the beats of the heart are audible is in the direct ratio of the feebleness and thinness of its walls." So far is this from being perfectly true, that I have met with cases in which the heart was dilated and attenuated to the extreme, yet the first sound was feeble. Since broaching this opinion in the first edition, innumerable observations have assured me of its accuracy. Nor should we expect it to be otherwise in such cases; for, when the heart, from extreme dilatation, is too feeble to contract smartly, its sounds must necessarily be weak. Hence they are so in ramollissement, and in the moments preceding dissolution.

Resonance on Percussion.—The resonance of the præcordial region on percussion is diminished by dilatation. The dulness is situated rather lower down than natural, and, as it is always in proportion to the increase of volume of the heart, it is greatest in hypertrophy with dilatation. When it extends over the inferior part of the sternum, it denotes dilatation of the right ventricle.

Dulness of the præcordial region on percussion may exist independent of enlargement of the heart: namely, when the anterior borders of the lungs are hepatized, and extend in front of the heart. I have met with a case of this kind in which the hepatized borders, forced completely over the heart by emphysema of the posterior parts, not only caused defective resonance, but prevented the impulse of an enormously hypertrophous heart from being perceptible. This, however, is a rare source of fallacy. On the contrary, dilatation may not occasion deficient resonance when the lungs are emphysematous, and their anterior margins are forced between the organ and the sternum; but this source of fallacy may be in a great measure removed by making percussion while the patient inclines forward and makes a complete expiration, by which the lungs are withdrawn, and the heart allowed to gravitate forward. I have seen the heart depressed into the epigastrium by great emphysema. I have also seen it thrown over to the opposite side of the sternum when the emphysema was confined to the left lung—especially if the right was condensed and contracted.

Physical Signs of Dilatation of the Auricles.—Auscultation has not hitherto supplied any *direct* signs of dilatation of the auricles; but as this affection is, in general, the consequence of disease of the valves, and of enlargement of the ventricles impeding the circulation through the heart, its existence may be inferred from the physical signs of these affections. Thus, when there is a contracted, and, still more, a permanently open state of either auriculo-ventricular orifice, dilatation of the corresponding auricle is almost certain: and when there is hypertrophy and dilatation of the right ventricle with much jugular congestion, dilatation of the right auricle is highly probable.

SECTION V.

PROGRESS, TERMINATIONS AND PROGNOSIS OF DILATATION.

In many persons the heart, without being dilated, has naturally thin walls; that is to say, (to assume a standard of comparison for an object which cannot have any fixed one,) the walls of the left ventricle are not, at the utmost, more than twice the thickness of those of the right. This state presents signs similar to those of dilatation, but in a less degree; namely, the impulse is diminished, the first sound is short and clear, and both sounds are more extensively audible than natural. Individuals so affected may live for a great number of years, even to an extreme old age, in a state of tolerably good health: it is only to be remarked that this conformation is in general accompanied with a delicate constitution, a slim stature, and small muscles. In fevers and diseases of the respiratory organs, the individuals in question experience, cæteris paribus, greater dyspnœa than others. If such a conformation augments, even slightly, a dilatation of the heart is the result.

A slight degree of dilatation is not a very formidable affection. The dyspnœa is sometimes not so great as to deserve the name of *morbid;* but the patient has simply a shorter respiration than most men, he more readily loses breath, and he experiences palpitations from much slighter causes. With these slight symptoms, however, he generally exhibits some delicacy of general health, and often presents a sallow, cachectic appearance.

This state (which is that of a great number of *asthmatics*) may subsist very long without occasioning any disorder of a serious nature; it may remain without making progress for a great number of years, and it does not always prevent the patient from attaining an extreme old age.

When dilatation has advanced so far as to occasion *morbid* dyspnœa, it has a constant tendency to increase, unless the circulation be kept tranquil by a very quiet life and judicious medical treatment, when necessary. With these precautions, the disease may commonly be cured; and, when not, it may generally be kept stationary, sometimes for an indefinite period, if not exasperated by fevers, inflammations, dyspepsia, or other affections, which, by hurrying the circulation, are eminently prejudicial.

When dropsy comes on, and, after having been removed by remedies, constantly shows a disposition to return, we may know that the dilatation tends to its fatal termination; and although the patient may sometimes rally from five, six, or even more attacks, he generally sinks in the course of one or two years, or less. The progress of dilatation with hypertrophy is much more rapid, as already explained in the chapter on hypertrophy.

Prognosis.—The general prognosis is founded on the above considerations, and is favourable so far as life is immediately concerned. The particular prognosis depends upon the degree of severity of the symptoms and the constitution of the patient. Dilatation with attenuation, and especially with softening, is the most destructive form.

SECTION VI.

TREATMENT OF DILATATION.

The treatment of dilatation with increased power of the heart, that is, with hypertrophy, is described in the chapter on hypertrophy. In this place I have only to speak of the treatment of dilatation with diminished power, that is, with attenuation, and sometimes with a natural thickness of the parietes.

The first indication is, to remove, if possible, the exciting cause of the dilatation; and if this be done before the disease has proceeded to such an extent as entirely to deprive the muscular fibre of its resilience and elasticity, these faculties come into operation and restore the organ to its natural size. Accordingly, if the cause be an obstruction in the pulmonary circulation, as that produced by hydrothorax, chronic bronchitis, emphysema, asthma, the use of wind-instruments, ventriloquism, &c., the attention must be primarily directed to the removal of these affections and the prohibition of these habits. If the cause be too violent exercises, mental emotions, inebriety, dissipation, occupations which, by placing the patient in a constrained posture, prevent the free circulation of the blood, as the professions of shoemaker or tailor, &c., the pernicious exercises, habits or professions must be abandoned and the mind calmed.

All the causes enumerated being of a temporary nature, the dilatation resulting from them, if not inveterate, can generally be removed. Of this I feel assured from careful observation during the last fifteen years, though I am aware that Laennec and many others regard the disease as incurable. But when the cause is permanent, as the contraction of an orifice of the heart, or a natural or acquired and long-established feebleness of the organ in proportion to its function, a complete cure of the dilatation is scarcely to be expected; but it may often be diminished, or kept stationary, and the life of the patient may sometimes be prolonged even to its extreme limits. In such cases, therefore, the practitioner should steadily and perseveringly pursue a palliative and prophylactic treatment, having first discarded from his mind the impression, no less erroneous in itself than detrimental to the progress of medical science, that organic diseases of the heart are necessarily fatal, and that therefore all treatment is unavailing.

The circulation should be kept as tranquil as possible by a strictly quiet life, and a moderate, unstimulating diet. The food, however, should be nutritious, comprising slightly under-dressed animal food, principally mutton and beef, twice a day, at breakfast and dinner, in order to keep the muscular system in general, and that of the heart in particular, in good tone. The same object may be promoted by a clear, dry, bracing air, as that of Brighton, and the shower-bath; from both of which I have seen the best effects result. Neither of them, however, have I found to suit those patients who have great pulmonary congestion with copious expectoration; as such require a warm, humid atmosphere to favour expectoration and the cutaneous function, and they cannot bear the shower-bath, on account of its determining too much from the surface to the heart and great vessels. Neither do they well bear opiates; as these remedies partly occasion diminished mucous secretion, and partly, accumulation of that already secreted; both of which circumstances increase the dyspnœa.

The general health and strength may likewise be improved by the occasional exhibition of bitters, mineral acids, and chalybeates, with aromatics. The preparations of iron in full doses, and in courses of from four to six weeks, with aloetic aperients and animal diet, are imperatively required and singularly beneficial if an anæmic state prevails. The stomach should be kept in good order; as its derangements—even a little flatulence or acidity, have a surprising effect in disturbing the action of the heart. The same may be said of the biliary secretion. When there are hysterical symptoms, antispasmodics, particularly the pilula galbani composita, and valerian, are very useful adjuncts to other remedies, due attention being also paid to the catamenia. If there be much nervous excitability with palpitation, sedatives, as digitalis, hyoscyamus, &c. may be employed.

Febrile and inflammatory affections of every kind, but particularly inflammation of the lungs and bronchia, should be sedulously guarded against, and, when occurring, should be promptly treated. Even a slight pulmonary catarrh should be viewed as a serious affection. To prevent colds, and relieve the heart by keeping up the circulation on the surface, flannel next to the skin is almost indispensable; and if the patient be chilly, as is frequently the case in dilatation, a jacket of wash-leather should be worn over the flannel during the winter. In short, the patient should be so clothed as to prevent chilliness, both within doors and out.

Attacks of dyspnœa are best relieved by immersing all the extremities in warm water, a blanket being thrown round the patient to promote perspiration, and fresh cool air being admitted to satisfy the craving for breath. While this is being done, he should take an antispasmodic draught composed of æther, laudanum, camphor, ammonia and assafœtida, combined according to circumstances.* It may be repeated two or three times, at intervals of from half an hour to an hour, according to circumstances.

Blood-letting should not be resorted to in dilatation with deficient power of the heart, *during the paroxysm*, and merely for the purpose of relieving it. The abstraction of a small quantity has not the effect, and that of a large is inadmissible, as it does more injury by increasing the debility of the heart, than it does good by lightening the circulation. Consequently, an ultimate aggravation of dyspnœa is the result. More than once, I have seen a large and indiscreet blood-letting fatal; as the patient could not rally from the exhaustion produced by the attack of dyspnœa to which that from the depletion has been superadded. If there be an absolute necessity for blood-letting, that is, if the dyspnœa be constant, and cannot be relieved by any other means, the quantity drawn should not exceed six ounces at one time, and it should be drawn very slowly, and during the intervals or remissions of the fits. In this way the bleeding may be repeated, if necessary, every one, two or three months, *provided it does not diminish, but, rather, increases the strength of the patient.* It must, however, be clearly understood that bleeding does not properly constitute a part of the treatment for dilatation with diminished power, but is an exception to the general rule.

For the treatment of dropsy, cough, &c., I refer the reader to the chapter on diseases of the valves.

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* Vid. for particulars, Treatment of Disease of the Valves.

CHAPTER III.

PARTIAL DILATATION OR REAL ANEURISM OF THE HEART.

The heart may be affected with real aneurism. In a young negro, who died suffocated, Corvisart found the left ventricle surmounted by a tumour almost as voluminous as the ventricle itself, containing several layers of rather dense lymph perfectly similar to those of aneurism of the limbs, and communicating with the cavity of the ventricle by a narrow, smooth, and polished aperture (Essai sur les Maladies du Cœur, p. 283). M. Bérard has recorded two similar cases, except that the tumours were only as large as ducks' eggs. In one, a portion of the sac was formed by the pericardium and fibrinous layers within, the muscular substance being entirely deficient. The general aspect of one of these preparations, examined by Laennec, led him to believe that aneurisms of this kind result from ulcerations of the internal surface of the ventricles. It will presently appear that this opinion is only partially correct. Four or five cases of the disease have occurred to myself. In one, (Brown,) steatomatous degeneration had caused the formation of a canal from the aorta, underneath one of the sigmoid valves and the internal membrane of the left ventricle, leading to an aneurism, as large as a nut, in the substance of the auriculo-ventricular septum. A similar case occurred subsequently in St. George's Hospital. A third, in which the aneurism was ossified, is delineated in Fig. 20. In the second case, the second sound was accompanied with a bellows-murmur. In the first and third cases, the physical signs were not noticed; yet there must necessarily have been a murmur with the second sound from regurgitation into the left ventricle, and that such was actually the case in Brown, is almost proved by the *jerking* pulse,—this state being invariably present in considerable aortic regurgitation. The general signs, in all the cases, were those of organic disease of the heart.

Mr. Thurnam has published a valuable paper in the Medico-Chirurg. Trans. vol. xxi, 1838, in which he has collected together seventy-four cases, thirteen of which, from the London and Chatham Museums, had not previously been described.

In fifty-eight of the seventy-four, the disease was situated in the left ventricle. In no instance has it been found in the right; which is apparently attributable to this ventricle being less subject to inflammation, and to great distensive pressure.

The following is an abstract of Mr. Thurnam's history of the fifty-eight cases in the left ventricle, to which he applies "the numerical method as rigorously as may be."

Lateral Aneurism of the Left Ventricle. "Lateral aneurism of the left ventricle is met with under two principal forms. Thus it may be either unattended by any external deformity of the heart, and confined altogether to the ventricular walls; or it may present itself in the form of a tumour growing from the exterior of the organ, and in size varying from that of a nut to that of the heart itself. In sixty-seven aneurisms occurring in the fifty-eight cases, thirty-five were attended by tumour; in nineteen there was no tumour: and in the remaining thirteen, it is doubtful whether tumour existed or not; although, from the small size of the sacs in these latter cases, it is probable that the disease scarcely extended beyond the surface of the ventricle."

"The size of the aneurismal sacs varies greatly: thus, in nine cases, their size might be compared to that of nuts; in twenty, to that of walnuts; in seven, to fowls' eggs; in fourteen, to oranges; and in nine cases, it almost or quite equalled that of the healthy heart itself. In one of these last named cases, the tumour had nearly presented externally. When the disease has been of some standing, and the sac has attained to a certain size, it usually opens into the ventricle by a mouth, the diameter of which is narrow, relatively to that of the sac itself; and the lips of which, like those of old arterial aneurisms, are generally projecting, well defined, and formed of a dense fibrous tissue. This kind of opening to the sac was present in at least twenty-five of the cases; whilst in nineteen others, which were mostly incipient, the mouths were as wide or wider than any other part of the sac, and no such projecting lips existed.

"With respect to the tissues of the heart engaged in the formation of the aneurismal sac, a careful analysis of the cases would seem to show, that in fifteen, the sacs were formed by the muscular fibres and pericardium; in four, by the endocardium and pericardium only; in twenty-five, by all of the structures entering into the composition of the walls of the heart; whilst, in twenty-three cases, the disease was either too far advanced, or the data are insufficient, to enable us to assign them to their proper places. The aneurismal sacs had in some cases undergone changes and transformations of different kinds; thus, in two cases, they are stated to have assumed a steatomatous structure; in three, a cartilaginous one; which latter change, in six others, was combined with a more or less advanced calcareous or osseous degeneration.

"In twenty-one cases, and probably in a still greater number, the sac had become strengthened by adhesion to the loose or fibrous layer of the pericardium; and in all these instances, the disease had advanced to the extent of producing tumour on the external surface of the heart."

"In six cases, in none of which had adhesion taken place between the aneurismal portion of the heart and the pericardium, and in which the aneurism scarcely, if at all, projected beyond the surface of the ventricle, a rupture of the sac had occurred, which had led to a fatal extravasation of blood into the pericardium. In one case only does rupture appear to have occurred when there was the adhesion alluded to, and in this instance the left pleura was the seat of the hæmorrhage."

"As regards the contents of the sacs, in twenty-three cases, which were chiefly those furnished with constricted mouths, and which were of considerable size, there was found a greater or less quantity of laminated coagula; seventeen, either apparently of less standing, or situated more in the direct channel of the blood, contained simple amorphous coagula; whilst nineteen appear to have been found empty after death. In three other cases, the contents were, in one, a hollow globular coagulum; in two others, simple fibrinous ones, evidently of old date.

"It would appear that no part of the left ventricle is exempt from becoming the seat of aneurism. Although a more extended acquaintance with cases than was possessed by M. Breschet at the time when he wrote on this subject, shows that this author was in error when he supposed the disease to be nearly, if not quite confined to the apex of the ventricle, yet this would still appear to be its *most* frequent situation. Thus the sixty-seven aneurisms which occurred in the fifty-eight cases, omitting one case in which this is not mentioned, may, as regards situation, be thus distributed; at or near the apex of the ventricle, twenty-seven; in different points of the base, twenty-one; in intermediate portions of the lateral walls, fifteen; in the interventricular septum, three. Of the cases in which the sac was seated at the base, four, which occurred to Dr. Hope, are remarkable from having opened both into the ventricle and into the aorta. Dr. Hope is of opinion that "steatomatous degeneration had caused the formation of a canal from the aorta underneath one of the sigmoid valves and the internal membrane of the left ventricle," and that, in this way, an aneurism had originated, which had ultimately opened into the cavity of the heart. It would, however, appear to me more probable, that the aneurisms had originally been formed in the ventricle, and had subsequently communicated with the aorta, as a consequence of the co-existent disease of the valves of that vessel; and I may observe, that this view would appear to be supported by four other cases in which the sacs had precisely the same situation, but in which there was no communication with the aorta. In the last of these cases, the preparation of which is in the museum at St. Bartholomew's Hospital, the contiguity of the aneurism to the aorta is such as to have led to its being described in the MS. catalogue as an aneurism of that vessel. Of the three cases in which the aneurism had its seat in the septum of the ventricles, one is only briefly alluded to by M. Cruveilhier as occupying its lower half, and as threatening to burst into the right ventricle. In another of these cases, recorded by Laennec, an accidental ulcerated canal had been formed in the highest part of the septum, and was accompanied by what would appear to have been a minute aneurism, containing fibrinous coagula. It is well known to anatomists, that the highest part of the septum, which occupies the angle between the posterior and right aortic valves, and which, in some instances of congenital malformation, is deficient, is in the human subject formed not of muscular fibres, but simply of the endocardium of the right and left ventricles almost in apposition, and strengthened only by the interposition of a little fibrous tissue continuous with that of the aorta."

"To conclude these observations on the situation of the aneurism of the left ventricle, the only general conclusion that we can come to appears to be, that the thinnest parts of its walls, or the apex and the highest part of the base, are those which are much more frequently than any others the seat of the disease.

"In general, or in fifty-two out of the fifty-eight cases, only one aneurism existed in each; but in four cases, two were met with in each: in one, there were three; and in another, four incipient aneurisms. In two instances, it is not improbable that two sacs, which were originally distinct, had coalesced, so as to form a single aneurism; and in another case, three sacs appear to have united in this way.

"An important point in the history of lateral aneurism of the heart, is that which relates to the other lesions of this organ, which are found to accompany it. To begin with the pericardium: in addition to the twenty cases already alluded to in which there was adhesion to the surface of the aneurismal tumour, we find that, in seven cases, there was general adhesion of this membrane to the surface of the heart; that in one, there was recent hæmorrhagic pericarditis; and that in three, there was dropsy of this cavity. In twelve cases, the endocardium is stated to have undergone different changes of structure; so as to have become either white, opaque, or thickened in the immediate neighbourhood of the sacs, or even more extensively; and in one case, there was a minute deposit of calcareous matter either in or beneath this membrane. The muscular substance of the ventricle was, in at the least nine cases, the seat of more or less extensive fibro-cellular degeneration, which was generally most marked around the sacs: in one case, there was a cartilaginous transformation; and in another, induration from a non-specified cause. In one instance, the walls of the ventricles are said to have been the seat of "lardaceous tumours," and in another, of extensively diffused suppuration. In numerous cases, there was a marked atrophy either of the fleshy columns which form the pillars of the mitral valve, or of the smaller ones which constitute the network on the internal surface of the ventricle. The valves of the left cavities are stated to have been diseased in ten cases; in five of these the mitral valve was the seat of the lesion, and was constricted by cartilaginous or osseous deposit; in three, the aortic valves were diseased, and both these sets of valves were implicated in one example. In eight cases, the valves are reported to have been healthy; whilst, in the remainder, their condition is not mentioned.

"Respecting the pathological changes in the heart, which we have thus seen to accompany lateral aneurism of that organ, it appears important to observe that they may almost universally be regarded as the effects of inflammation. With respect to a majority of them, or those seated in the muscular tissue and pericardium, there can, on this head, scarcely be a doubt; and although some difference of opinion may still exist respecting the alterations which have been alluded to as involving the endocardium and the valves, yet they are now very generally, and I think correctly, regarded as the consequences of inflammation. From this part of our inquiry also, I think we can scarcely avoid drawing the inference, that aneurism of the heart cannot be regarded as exclusively dependent upon pathological changes in one only of the tissues entering into the composition of this organ."

"The number of cases in which the heart is not stated to have been the subject of some lesion, (hypertrophy, dilatation, &c.,) in addition to the aneurism, does not exceed ten; and in three only is it positively stated to have been otherwise healthy."

As respects the influence of sex; in forty cases, in which this is recorded, thirty occurred in males, and ten in females. The proportion thus met with in the female, is much greater than is found to be the case in arterial aneurisms, which, according to Hodgson, occur eight, and according to Lisfranc, eleven times oftener in the male than in the female. Even as respects aneurism of the aorta, the most common variety of the disease in the female, Dr. Hope has only found the proportion to be rather larger than that indicated by Hodgson.

"The age of the patient is either stated, or to be inferred with tolerable accuracy, in thirty-five cases."

"It appears, that after adult age, cardiac aneurism is not remarkably confined to any particular period: although it would seem to prevail with the greatest frequency at two distinct periods, or between the ages of twenty and thirty, and again in very advanced life. In this respect, then, we likewise find that cardiac aneurism differs remarkably from arterial, which, according to the experience of Sir Astley Cooper, and also from an analysis of 108 cases by M. Bizot,* prevails chiefly between the ages of thirty and fifty."

"There can scarcely be a doubt, that, as of other organic diseases of the heart, so also of aneurism,—acute rheumatism, affecting this organ, either in the form of endocarditis or of pericarditis, is to be regarded as closely connected with the production, if not as the efficient cause, of this lesion. If this view should prove to be correct, we shall have no difficulty in explaining the greater frequency of cardiac than of arterial aneurism during early life; as it is well known, that in the progress of acute rheumatism, the inflammatory affections of the heart which have been alluded to, occur much oftener at this than at any other period.

"The exciting cause of the disease would appear to have been external violence, in the form of an injury of the chest, in the case of the gondolier, a fit of violent anger in that of the nobleman, protracted mental anxiety in another instance, severe efforts on the stage in the character of Hamlet, in the case of Talma, and in a fifth instance, the retention of the breath during a military flogging.

"From an examination of the anatomical details, as well as of the apparent causes of the disease, in reference to the determining of its nature, I come to the conclusion, that in twenty-two cases out of the fifty-eight, the aneurism originated in a dilatation of all the structures entering into the composition of the walls of the heart; and in six, in a solution of continuity of the lining membrane and inner stratum of muscular fibres, either as a consequence of ulceration, or, what is more probable, of rupture; whilst, in the remaining thirty cases, the disease was either too far advanced, or the data given are insufficient to enable us to form a satisfactory opinion on this question.

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* Mém. de la Soc. Méd. d'Obs. tom. i. p. 409. Recherches sur le Cœur et le Système Arteriel.

"I therefore conclude that this lesion, in by far the greater proportion of cases, is of the nature of *true aneurism:* or that it has its origin in the dilatation of a portion of the walls of the heart, which has become less able to resist the distending force of the blood during the ventricular systole, in consequence of organic changes in the tissues composing it. These changes may be confined to one of these tissues, as the endocardium; or they may involve that membrane and the muscular structure simultaneously; or, lastly, they may, I believe, originate in the pericardium, and be propagated from without, inwards. In a great majority of instances, these changes would appear to have been the result of a more or less active antecedent inflammation."

"In the departments of symptomatology and diagnosis, the information that we possess relative to this form of disease is less extensive and precise than that relative to its pathology. It is probable that, in its incipient forms, aneurism of the heart is not necessarily attended by any derangement in the function of this organ. In two cases, it is expressly stated that no symptoms referable to the heart existed during life, and in these the disease was in a very early stage.

"The mode of incursion of the disease differs remarkably in two classes of cases. Thus in three instances the attack was sudden, and attended with marked symptoms, analogous to those observed in cases of rupture of the heart, when this is not directly fatal. The most instructive of these cases is that of the nobleman, related by Galeati, who, after a violent fit of anger, was suddenly seized with severe præcordial pain, orthopnœa, agitation, fear of death, a disposition to syncope, and a vibratory, frequent, but languid pulse.*

"In cases of this description, the mode of attack, as well as the immediate causes, would lead us to conclude that the disease is of the nature of false aneurism from rupture.

"In the great majority of cases, however, the disease would seem to have had a very insidious origin, and to have been only very gradually announced by symptoms. This is what we should be prepared to expect in cases of true aneurism; and it may be observed that this difference in the mode of attack in the two forms of the disease, corresponds with, and supports the conclusion, which, chiefly on anatomical grounds, has been already come to, that true aneurism of the heart is much more common than false.

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* I have noticed the same symptoms in six or seven cases of rupture of the aorta or of valves (see p. 199).

"In five cases, the symptoms of the disease are described generically as those of "diseased heart." In twenty-three cases in which the symptoms are given in detail, these, taken in the order of their frequency, were as follow; dyspnœa, in several instances amounting to the severest form of orthopnœa, in fifteen cases; præcordial pain of different characters, in one or two cases amounting merely to uneasiness, but in several others accompanied by a sense of weight, in fourteen; dropsy more or less extensive, in ten cases; palpitation in nine cases; anxiety, dread of death, or restlessness, in eight; and syncope, or a disposition to it, in three cases.

"In addition to these symptoms others are also more rarely mentioned; such as cough, throbbing of the carotid arteries, pulsation of the jugular veins, livid or blue countenance, and hæmorrhage from the nose and lungs. The condition of the pulse is noted only in a few of the cases, and in seven of these it is stated to have been feeble, sometimes in an extreme degree."

"As aneurism of the heart has seldom been met with, uncomplicated with other lesions of this organ, great difficulty necessarily attends our forming any conclusions as to the general symptoms, if any, which distinguish it."

"But in addition to the class of symptoms now alluded to, a variety of distressing sensations in the præcordial region were experienced in a great proportion of cases; which would, to a certain extent, serve to distinguish the disease from cases of simple dilatation with or without hypertrophy. Symptoms of this description are met with in two well-known forms of disease of the heart, angina pectoris and valvular disease, and especially in those cases in which there is much ossification. Now, although the group of symptoms which are known by the name of angina pectoris, cannot always be referred to an ossified and indurated condition of the coronary arteries, valves, and origins of the great vessels, yet as the best recent authorities concur in the belief that, in the majority of cases, it is associated with these or similar changes, we shall not perhaps be generalising too rapidly, in ascribing the uneasy sensations and pain, which are met with in these three forms of disease, to one common cause.

"This would appear to me to consist in the irritation occasioned by the rigid and inelastic morbid structures to the cardiac nerves, and especially to those derived from the great pneumogastric,—a nerve which recent experimental researches, and especially those of M. Brachet, as well as anatomical analogy, have shown to be, to a certain extent, in all probability, a sentient nerve. The character of the pain experienced in aneurism of the heart differs, as we have seen, in different cases, but in some it was described as being accompanied by a peculiar and distressing sense of weight; a kind of pain more intense, but still very similar to that often complained of in aneurism of the thoracic aorta.

"The diagnosis of aneurism of the heart must, in the present state of our knowledge, necessarily remain very doubtful. Indeed, it is not to be expected that a diagnosis will ever be effected, without the aid of the information to be derived from an acoustic and manual examination. Unfortunately, the cases in which the physical signs have been observed are very few in number. In three cases, the impulse of the left ventricle is stated to have been increased; in one, the action of the heart generally was forcible and tumultuous; and in two others, feeble and obscure. In four cases, a bellows or rasping sound was heard with the ventricular systole; and in a fifth case, a similar sound was heard to the left of the sternum. In one case, the character of the first sound was short, like that of the second."

Such is the substance of Mr. Thurnam's account of real aneurism of the left ventricle. The signs, as he admits, are wholly insufficient to render the affection distinguishable from ordinary disease of the heart. The physical signs, in particular, are too imperfectly noticed to indicate anything. So long, indeed, as particular valvular diagnosis was impossible, it was not to be expected that murmurs could indicate anything more than some valvular affection. But now, when particular valvular diagnosis is practicable, it remains to be seen whether a new series of accurate observations will not, by excluding valvular disease, leave distinctive signs of real aneurism. The mode of procedure would be this.

If a given murmur was not amenable to the rules explained under the *physical signs of valvular disease*, nor to those of the attrition-murmurs of pericarditis, (p. 106,) it might be presumed to indicate something anomalous or new. The presumption would be strengthened if the pulse was also irreconcilable with the rules applicable to the individual valvular diseases (See *Pulse of Valvular Dis.*). Accurate notes should also be taken of any anomaly in the nature, situation, and synchronism of the impulse, and in the situation and extent of dulness on percussion, not referable to ordinary enlargement of the heart, or to fluid within the pericardium; for a small proportion of real aneurisms form tumours exterior to the heart of such magnitude, as possibly to be capable of producing the anomalies in question. Finally, the heart should be examined after death, and a code of distinctive rules should thus gradually be worked out. It was by a procedure of this kind that I came to the conclusion, in the case of Mitchell, that there was something extraordinary or new. The event proved the existence of an aneurism bursting out of the aorta into the right ventricle; and I have drawn out rules, which will probably distinguish this affection for the future. To offer another exemplification—I could suppose that, in the case delineated in Fig. 20, if the physical signs had been noticed, the murmur of aortic regurgitation, instead of gradually diminishing down the ventricle, would have been as loud or louder in the middle and lower parts of the cavity, because the aneurism opened in that situation: while, at the same time, I should have felt assured that the murmur did not result from mitral contraction, for two reasons: first, that there was the jerking pulse of aortic regurgitation; and secondly, that there was *not* the small, weak, irregular, intermittent, and unequal pulse of mitral contraction, nor a murmur with the first sound indicating mitral regurgitation. Under such circumstances, then, something peculiar might have been inferred, and a real aneurism might have been assigned as one of the presumptive causes of the anomaly.

On the whole, I am not very sanguine respecting the possibility of detecting many of the partial aneurisms; as a large proportion are so small, and so situated, as probably not to create any signs whatever; and many others, implicating the valves, would probably occasion nothing more than the ordinary signs of valvular disease.

We now proceed to Mr. Thurnam's account of true aneurisms of the auricles.

"*Aneurism of the Auricles.*—The number of cases of aneurismal dilatation which are recorded as having occurred in the left auricle, is much less than that which we have seen to be the case in the ventricle. The disease would appear to have been nearly uniformly of the diffused kind, and to have generally involved the entire sinus of the auricle."

"In one case only with which I am acquainted, was the aneurism of that circumscribed kind to which the term *lateral* or *sacculated* could be applied. In this case there was a sac as big as a nut hanging over the base of the left ventricle, and containing dense fibrinous concretions and liquid blood, which communicated with the cavity of the auricle by a canaliculated pedicle an inch in length."

"In all the cases with which I am acquainted, whether occurring in the sinus or appendage of the auricle, and which are nine in number, the disease was connected with, and appears to have been dependent upon, an extreme contraction of the mitral orifice, producing a difficult transmission of the blood from the left auricle."

CHAPTER IV.

SOFTENING OF THE HEART.

Softening of the heart presents the following anatomical characters. The organ, when placed on a table, does not maintain its round form, but sinks and becomes flattened. When the ventricles are opened by an incision, they collapse, even though thickened. The muscular substance feels flaccid, and tears with great facility. Sometimes it is so soft and friable, as easily to break up under slight pressure of the fingers. These characters are common to all the varieties of softening.

Before noticing the varieties, it may be premised that M. Laennec and M.M. Bertin and Bouillaud differ in their opinions as to the nature and cause of softening. Laennec supposes it to be "an affection *sui generis*, resulting from a derangement of nutrition, by which the solid elements of the tissue diminish in proportion as the liquid or demi-liquid elements augment. All the muscles soften in a moderate degree in a great number of diseases, acute and chronic: a few days suffice to produce this effect . . . . and the change takes place without any sign of inflammation." M. Bouillaud, (who, in his conjoint work with M. Bertin, was the sole author of all the doctrines relative to inflammation—a fact which M. Laennec states (Traité, ii. p. 538, note,) to have been personally communicated to him by M. Bertin,) contends that softening, in all its varieties of colour, is a result of inflammation; because, as "softening of the brain, the uterus, the kidneys, the spleen, &c. is, in the present day, regarded as a certain characteristic of inflammation," he could not adopt another opinion without doing violence to the laws of analogy.

According to my observation, both of these conflicting opinions are partly correct, and partly erroneous from being too limited.* The reasons for this view will become apparent as we proceed.

Softening is almost invariably accompanied with some change of colour, and Laennec has noticed three varieties: 1st. *red*; 2d. *yellow*; 3d. *whitish*. M.M. Bertin and Bouillaud, and more recently M. Bouillaud, appear to have recognised the same. As the arrangement by colour is convenient, I shall continue, as in the original edition, to follow it.

According to my observation, each of the varieties may be inflammatory or not. When inflammatory, the *red* corresponds with the first stage of carditis, and is analogous to the inflammatory engorgement constituting the first degree of peripneumony; the *whitish* corresponds with a more advanced stage, analogous to the second and third degrees of peripneumony, when a pale tint is produced by the absorption of the red particles of the blood, and by the presence of lymph and pus in variable proportions. The *yellow* variety is more, I think, a result of chronic inflammation. I shall now proceed to describe each of these varieties, and shall point out, in passing, the circumstances under which each may exist independent of inflammation.

1. *Red Softening.* This variety presents a claret, morone, or violet colour, denoting an excess of blood in the muscular substance, and I have occasionally seen it speckled, as if with extravasated blood. That softening with increased redness may result from acute inflammation, rests on incontestable evidence; for it was found by Dr. Latham to exist in a remarkable case in

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* I find that M. Bouillaud, in his more recent work in 1835, has modified his previous opinion, and espoused that maintained in the text. "We must not be astonished," says he, "at the confusion which reigns in what M. Laennec has said on the subject, because, under the generic term of softening, he has confounded different morbid states. This confusion has proceeded to the extent of assigning the same name to mere flaccidity of tissues, (mollesse,) and to softening (ramollissement) such as I here describe it (viz. from carditis); *it must not be concluded that, because I have considered carditis capable of producing softening of the heart, I regarded every species of softness or softening as a result of inflammation* (Traité, ii. 294; note). Again he says, (p. 296, note,) "It is possible that the softening in question (viz. the yellow) is met with in a slight degree in subjects who had not presented any symptoms whatever of obscure and chronic carditis: such are certain cachectic individuals. This is an important question to examine."

which, says he, "the whole heart was deeply tinged with dark-coloured blood, and its substance softened; and here and there, upon the section of both ventricles, innumerable small points of pus oozed from among the muscular fibres. This was the result of a most rapid and acute inflammation, in which death took place after an illness of only two days." The phenomenon of pus infiltrated throughout the whole muscular substance of the heart had never been known to have occurred when Laennec wrote, as appears from the admission of that author himself; and it nullifies one of the strongest arguments by which he supports his view that softening is not inflammatory: namely, "I think," says he, "we may regard it as a general law in the economy that all the *soft* tissues *harden* by the effect of a *true* inflammation, that is to say, *tending to the formation of pus*"—this being the only definition of inflammation that he admits (De l'Auscult. ii. p. 541). Now, it is proved by Dr. Latham's case just cited, that inflammation, taking his own definition of it—namely, "inflammation tending to the formation of pus," and actually forming pus, is capable of producing softening, with increased redness: we may, therefore, without trangressing the laws of philosophic caution, presume that it may produce the same effect, thought *not* attended with the formation of pus; for every inflammation does not necessarily end in suppuration. This view is countenanced by the circumstances under which, according to the concurrent testimony of all parties, the species of softening in question is very frequently found; namely, accompanying acute pericarditis or endocarditis. I have delineated two well-marked specimens of this in my work on Morbid Anatomy, figs. 56 and 64, in neither of which was there any pus. Red softening from inflammation, in the instances in which I have witnessed it, has been attended with a higher degree of tenderness and lacerability than when not inflammatory.

It remains to be shown under what circumstances red softening is *not* inflammatory. I have frequently found it where there was a retardation of the venous circulation through the muscular substance; as in dilatation with attenuation, great obstruction of the mitral, and occasionally of the tricuspid valve, &c. Under these circumstances, however, yellow softening is also common. Again, red softening may result from a diseased, incoagulable state of the blood, as in scurvy, typhus fever, &c. M. Bouillaud, in his conjoint work with M. Bertin, ascribed the redness in typhus fever to inflammation.* Laennec, with better reason, refers it to the alteration or putrescence of the liquids, having always, as he states, found it greater in proportion as the alteration was more decided. This accords with my own observation. M. Louis also has found the same in typhus, especially when death took place rapidly, the pulse having been feeble, fluttering, irregular and hurried.†

Laennec inquires whether softening "could be the cause of the extraordinary frequency of the pulse which often supervenes during convalescence from fevers, and which sometimes persists for several weeks, though the patient regain strength and substance." Bouillaud resolves this question in the affirmative, but adds that the quickness of the heart's action can only be accounted for on the view that the softening is a genuine carditis. In his later work in 1835 he thinks that mere inflammation of the internal membrane is sufficient to produce the quickness of pulse in question (Traité, ii. 303). I do not see the necessity of resorting either to carditis or to endocarditis to account for the quick pulse. Softening independent of inflammation is sufficient to explain it, but there is another cause which may contribute to its production, or which may even alone produce it: I allude to the poor and attenuated state of the blood usually following typhus fever—the state, in short, of anæmia, which is amply sufficient to maintain the pulse at 100 to 120 per minute, until the gradual restoration of the colour of the patient evinces that the blood has regained its natural consistence and quantity.

2. *Whitish Softening.* This variety appears to have been only glanced at by Laennec. "It ordinarily accompanies pericarditis, says he, and is not observed in any other case . . . . it is accompanied with whitish paleness of the substance of the heart . . . it never proceeds to such a point as to produce friability of

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* In his later work, he still maintains that this may sometimes be the case, but he also admits softening from "putrid decomposition" (Traité, ii. 302; note (2)).

† Red softening from putrefaction subsequent to death must be carefully distinguished from real softening; and this may be accomplished by attending to the well known rule of not postponing the autopsy longer than twenty-four hours after death, especially in warm, damp weather. Even twenty-four hours are too long in cases of diseased blood, as typhus, scurvy, purpura, &c.

that substance: and often, the degree of consistence does not even appear to be sensibly diminished, although the organ have become flabby, and its walls completely sink after an incision" (De l'Auscult. ii. 535). M. Bouillaud thinks that this description applies to an identical affection more fully described by Corvisart in the following words, relating to a case in which pericarditis with effusion accompanied white softening of the heart: "Carditis ultimately converts the muscular part of the heart into a soft and pale substance; the fleshy fibres then retain little tenacity; the cellular tissue which unites them appears lax, sometimes it is charged (*pénétré*) with matter consisting of lymph nd pus (*lymphatico-purulente*); in certain cases, it is in part destroyed; the vascular system is more apparent, more developed, than in the natural state, and appears to participate in the inflammation of the other tissues. The walls of the heart tear with the least effort, and strong pressure is not necessary to reduce them to pulp" (Corvisart, p. 257).

This description, if correct, evidently applies to an advanced stage of acute carditis with effusion of lymph and pus, a state analogous, as already remarked, to the 2d and 3d stages of peripneumony. I have never seen an instance of the affection: nor does M. Bouillaud, so far as I can discover, directly state that he has been more fortunate: whence I conclude that the inflammatory whitish softening is rare, a circumstance which may perhaps be accounted for by supposing that the patient either dies or recovers before the inflammation attains so advanced a degree.

I have frequently met with a very pale and flabby condition of the heart in cases of great anæmia, especially with atrophy of the organ.

3. *Yellow Softening*. This variety, which is much more common than either the red or the white, presents a faint yellow or fawn-coloured tint, aptly compared by Laennec to that of the palest dead leaves, and it bespeaks a deficiency of blood. It may pervade the whole, or portions only of the heart, and may co-exist with hypertrophy, dilatation, or other lesions of the muscular substance.

I entertain no doubt that the yellow softening may, in some cases, result from inflammation, as I have seen it penetrate only a certain depth, for instance, two or three lines, into the muscular substance, as if propagated from the inflamed pericardium, which had either become adherent, or contained false membrane and fluid.* I have also seen the same yellowness penetrate a line or two from the internal membrane, which presented vestiges of endocarditis. I have likewise seen it pervade the whole thickness of the walls in patients who, at no very remote period, had laboured under pericarditis.

But though yellow softening may be inflammatory, I fully believe, with Laennec, that it may occur independent of inflammation in subjects who have long been in a cachectic state, or who have been worn down by slow anæmic marcor or hectic fever. For I have repeatedly met with instances of enlarged heart, in which the organ was universally pale, flaccid and somewhat lacerable, yet no inflammation or fever had antecedently existed to account for the state. The affection therefore appears to have been referable to the same causes as, in such cases, sometimes render the other muscles pale, flaccid and withered. Laennec remarks that those who are affected with this chronic yellow softening, "have a pale, sallow complexion, and a withered skin; and even when they are attacked with dilatation or hypertrophy, as almost always happens, they do not present any tumefaction and lividity of the face. Their lips are rarely violet and still more seldom bloated; on the contrary, they are almost always nearly colourless" (De l'Auscult. ii. 586). This statement is, for the most part, correct; for, in fact, such patients are generally anæmic: yet, in the case of individuals with naturally florid complexions, especially if of plethoric habit, the presence of anæmia does not prevent the cheeks, nose, and lips from becoming purple or livid, and the face and lips more or less bloated: the patients, in short, are amenable, though in a less degree, to the same general rules with respect to colour, as are explained at p. 267, in reference to hypertrophy.

As softening diminishes the cohesion, and therefore the elasticity of the heart, we are necessarily led to infer that it conduces to dilatation: accordingly we find that dilatation is its almost constant concomitant, when it has subsisted for a considerable period.

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* A specimen of this is delineated in fig. 61 of the writer's Morbid Anatomy.

Signs and Diagnosis of Softening.

General Signs. — As softening from acute inflammation is almost—perhaps wholly, unknown except as a concomitant of pericarditis or endocarditis, there is difficulty in distinguishing its signs from those of the other maladies. Complicated with them, it is attended by a quick, feeble, small, and faltering pulse, great anxiety, and a disposition to syncope—the same symptoms, in short, that characterise pericarditis with copious fluid effusion, and endocarditis with polypus choking the cavities. Now, as copious effusion or polypus is often present when the inflammation is so severe as to affect both the membranes and the muscular substance, it is scarcely possible, in every case, to say positively whether the severe symptoms in question depend on the effusion and polypi, or on the softening. I feel assured, however, that the latter, as well as the former, is capable of producing them; as they sometimes exist when there is neither polypus, nor a quantity of fluid sufficient to constitute an adequate cause, and as it is consistent with analogy to suppose that the muscular tissue of the heart, when softened by inflammation, would, like other muscles, be rendered incapable of adequately discharging its function. In this point of view, softening greatly aggravates the severity and danger of endocarditis and pericarditis.

The general symptoms of softening from chronic inflammation or other wasting disease, as scurvy, hectic, anæmia, typhus fever, &c., are no less ambiguous; as they may result from the primary disease itself, independent of softening. They are, general languor; a sallow, exsanguine, withered complexion; with a purple or livid tint of the cheeks and lips in the naturally florid; a quick, but soft and feeble beat of the heart and pulse, often with great intermittence, irregularity and inequality; gradual reduction of the strength; and dropsical effusion, sometimes amounting to general anasarca, from inability of the heart to propel its contents.

I have frequently found softening after a series of symptoms mentioned by Laennec: namely, when, in a case of dilatation with or without hypertrophy, there have been long and frequent attacks of suffocative dyspnœa; when the struggle between life and death has been protracted,—of several weeks' duration, for instance; and when the violet hue of the face, the extremities and the other parts of the surface of the body, had announced, long before death, the retardation of the blood in the capillary system. The three cases appended to the present chapter, were of this description. I have invariably found such cases attended, after death, with great pulmonary engorgement, and often with the "pulmonary apoplexy" of Laennec. Passive hæmoptysis of dark, grumous blood frequently exists during the last days of life.

Physical Signs.—As the systole and diastole of the heart are enfeebled by softening, its impulse is more or less reduced in strength; and it frequently happens that the beats are not only intermittent and irregular, but very unequal in force, an occasional beat being pretty strong, while the others are very feeble, or even imperceptible. When these occasional beats are *decidedly* stronger than natural, I always venture to found on them a diagnosis of hypertrophy. Both the sounds are rendered weaker than natural by softening, and the first sound becomes short and flapping like the second, in consequence, I presume, of its being produced solely by extension of the auricular valves, the ventricular systole being too feeble to generate muscular sound. This flapping character of the first sound, even though hypertrophy accompany the softening, has not hitherto been noticed by authors as characteristic of the latter affection.

Diagnosis.—In the former editions of this work, I experienced a difficulty in detecting softening when complicated with hypertrophy, because it could not be affirmed, that the diminution of the first sound was not referable to the hypertrophy rather than to the softening; but this difficulty is now removed by the first sound in hypertrophous softening being of the short, flapping character above noticed, while there still remain the ordinary physical signs of hypertrophy—the augmented impulse, either constantly or with occasional beats, and the increased extent of dulness on percussion. The irregularity of the pulse presently to be noticed, is an additional indication of softening, because this sign is foreign to mere hypertrophy.

The diagnosis of softening from disease of the valves, especially of the mitral, producing an irregular pulse, requires particular notice. When the first edition of this work was published, I was not sure that softening had any particular effect on the regularity of the heart's action: but I have subsequently ascertained that, when considerable, and especially if conjoined with dilatation or even hypertrophy with dilatation, it produces an eminently small, weak, intermittent, irregular and unequal pulse, such as is occasioned by the highest degrees of disease of the mitral valve. Three cases, selected from several others, are appended to the present chapter, expressly for the purpose of exemplifying this fact, which has hitherto been overlooked by authors.

But though the pulse of softening be the same as that of disease of the mitral valve, there is little difficulty in the diagnosis of the two affections. If, after an exploration made according to the rules offered at p. 90, no murmur be found to attend either sound of the heart, the irregularity of the pulse must be ascribed to softening, provided it be not referable to temporary nervousness, to a paroxysm of dyspnœa, or to ebbing of the vital powers on the approach of dissolution—all of which circumstances are capable of producing transitory weakness and irregularity of the pulse, even in a healthy heart.

Prognosis.—The prognosis of softening depends much upon the co-existent and, as it were, primitive affection. As above stated, it greatly augments the danger of pericarditis, and probably of fever; but when these affections terminate favourably, there is every reason to believe that the muscular substance may be restored to its healthy condition. With chronic maladies, and especially organic disease of the heart, softening is an aggravant of the worst kind; for it not only contributes powerfully to weaken the heart, but, by impairing the tone and elasticity of the muscular fibre, it has appeared to me to counteract that natural tendency of the heart to recover itself from dilatation, and dilatation with hypertrophy, which has been shown (p. 288) to exist in a high degree under favourable circumstances of treatment. Hence, of all cases of dilatation, those attended with softening are, cæteris paribus, the most difficult to cure.

Treatment. — When accompanied by acute inflammation, softening must be treated on the same principles as pericarditis. When a result of chronic disease, it demands the same remedies as the primary affection, and especially iron, bark, a nutritious animal diet, and good air, if they be not otherwise contraindicated. Iron in full doses is particularly required, and is eminently useful, in anæmic subjects. These remedies must be superadded to perfect tranquillity of body and mind, and the other means calculated to prevent palpitation and engorgement of the organ, as already explained at p. 317, under the head of Dilatation. Complete cessation of palpitation is not to be expected till anæmia is removed, for this alone is capable of maintaining the symptom. Nor is a diminution of co-existent dilatation to be looked for till a restoration of the tone of the general muscular system and a decrease of the physical signs of softening, denote that the heart has recovered somewhat of its natural tone and elasticity. If the treatment should commence at an advanced period of the disease when dropsy has set in, the diuretics employed should have a tonic basis, as cascarilla, quina and gentian; the strength should be carefully supported by as much animal nutriment as the stomach will bear; and diffusible stimulants—even wine and brandy, should be administered, if there be a decided failure of the circulation, with tendency to sinking, especially in the last stage of softening.

The three following cases illustrate softening.

Dilatation ; softening ; irregular pulse. No valvular disease.

Case I.—A. B., a man in St. George's Hospital, under Dr. Chambers, Aug. 12, 1835. Had been nearly drowned eight months previous to admission. Symptoms of disease of the heart ensued. I found very extensive dulness on percussion. *Impulse* slight, and very irregular; *Pulse* extremely irregular, unequal and intermittent, so that there was the greatest difficulty in ascertaining its coincidence with the ventricular systole, which was frequently unattended with pulse. *Sounds*, both very weak, and the first of a short, clicking character. No murmur. Was subject to agonising dyspnœa, and died during a paroxysm of three days duration. *Diagnosis.* Dilatation: no disease of the valves.

Autopsy (at which I was not present). Heart greatly enlarged by dilatation, with about the natural thickness of the walls, but they were *very soft and flabby*. No disease of the valves.

Remarks. This was one of the first cases which gave m [illegible] that soften-ing was [illegible] of irregularity of the heart's action. The following remarks are appended to the case in my note-book. "The softening appears to have been the cause of the extreme irregularity of the heart's action; as, when the muscular substance is firm, an equal degree of dilatation may be unattended with irregular pulse, except during paroxysms of dyspnœa, or failure of the vital powers. May not this irregularity, therefore, be added to the signs of softening when there is no disease of the valves, indicated by murmur, to account for the irregular pulse?"

On these grounds I gave a diagnosis of softening in the two following, amongst other cases. In all the cases, the patients died after a difficult and protracted struggle, as described by Laennec.

Softening. Dilatation. No valvular murmur; pulse extremely irregular. Great pulmonary apoplexy.

Case 2.—Mr. Wm. Summers, æt. 4[illegible], fat and plethoric. I attended him with Mr. Farquhar, surgeon, in January 1838. Ill a year. Now, purple and livid lips and cheeks; sallow between; *bloated*: great dyspnœa, but no orthopnœa: considerable anasarca; no hydrothorax. Impulse not perceptible. First sound as short and flapping as the second, and there are two or three *sounds* for every distinct beat of the pulse, the other beats being either very feeble or wholly imperceptible. Second sound, above the sigmoid valves, distinct but feeble. Dulness on percussion. Died in three weeks, without hæmoptysis.

Diagnosis. Dilatation; softening; no valvular contraction or regurgitation; no hydrothorax.

Autopsy. Very extensive *pulmonary apoplexy* in both lungs, which were, in consequence, much diminished in volume. No hydrothorax. *Heart* enlarged to double. Walls of *left ventricle* half an inch thick, and rather flabby and pale (softening); its cavity enlarged to double. Walls of *right ventricle* a quarter of an inch thick: cavity large. *Aortic valves* very slightly thickened, but not contracted: capable of discharging their function. *Mitral valve* also thick and opake, and chordæ tendineæ rather thick, but the valve admitted three fingers easily, and seemed capable of closing the orifice. *Tricuspid* and *pulmonic valves* rather thickened, but not contracted.

Remarks.—We here again see that a most intermittent, irregular, unequal, small, and weak pulse, with extreme venous retardation—symptoms usually supposed to be dependent on valvular disease, resulted from softening with dilatation alone. I inferred the softening from the state of the pulse being unattended with valvular murmurs, and from the weakness of the sounds. The dilatation was indicated by the flapping character of the first sound, the dulness on percussion, and the absence of impulse.

Extreme softening; great hypertrophy with dilatation; no valvular disease; pulse extremely irregular, &c.; pulmonary apoplexy.

Case 3.—Sir ——, Bart., whom I attended with Dr. Chambers, æt. 69, fat, fifteen stone. Had been affected for six or seven years with an intermittent pulse, and slight hurry of the respiration on exertion: three years before death, he had severe jaundice, and subsequently had slight ailments, which were ascribed to derangement of the stomach and liver. A fatty state of the heart being at length suspected, immoderate exercise was injudiciously taken, with the view of reducing it. He was attacked with constant palpitation, most oppressive dyspnœa, and complete sleeplessness. As these symptoms did not abate in a week, he travelled 100 miles up to London in one day, and on his arrival I saw him for the first time. His complexion was very sallow, with purplish cheeks, nose and lips; oppressive dyspnœa, but not orthopnœa; pulse extremely intermittent, irregular and unequal, an occasional beat being stronger and larger than natural, while the intermediate beats were very small, weak, and often imperceptible. Slight œdema pedum.

Auscultation. Impulse, a flutter with an occasional bound of inordinate force. *Sounds*, both weaker than natural, and the first as short and flapping as the second. No murmurs. Contractions of the heart were 130 to 140 per minute, and the pulse 40 to 60. *Percussion*, prevented by a vesication on the præcordial region.

The symptoms increased. Whenever drowsiness slackened voluntary respiration, gasping came on and aroused him, and this occurred alternately every four or five minutes. Next supervened expectoration of dark blood, failure of the pulse, and moderate anasarca: finally, diminution of sensibility, and death in a fortnight, after a protracted struggle.

Diagnosis.—Hypertrophy with dilatation, which I inferred from the occasional strong impulse and strong, large pulse. No valvular contraction or regurgitation, because no murmur, and because great mitral contraction or regurgitation was incompatible with the occasional strong beats of the pulse, which is always weak in such mitral disease. Softening, because, without mitral disease, the pulse was irregular, &c., and because there was passive hæmoptysis and great venous retardation; also because the sounds were weak and the first short, though there was hypertrophy with dilatation. Pulmonary apoplexy. No hydrothorax.

Autopsy.—Cavities of the pleura contained two ounces of bloody serum. *Lungs*, universally gorged and black: whole inferior lobe of the left, in the state of pulmonary apoplexy. *Heart*, dilated to the size of a bullock's, being nearly three times as large as the closed fist of the subject. Walls of left ventricle thickened to about three-quarters of an inch: muscular substance dark red from sanguineous engorgement, and so much softened that a finger and thumb passed through it with very little pressure. All the valves perfectly healthy and capable of discharging their functions, except that the mitral and aortic were strong and rather opake, from hypertrophy of the fibrous tissue. Walls of the right ventricle, of natural thickness, but the external third of the muscular substance was replaced, over a considerable extent, by fat. Auricles dilated to double.

Remarks.—This case presents the general signs usually ascribed to great valvular disease, and I adduce it, both to evince that softening alone may occasion these signs, and to furnish data for the diagnosis. Pulmonary apoplexy has not, I believe, been noticed as a consequence of softening. I have met with it in several other cases, as well as the present. It is produced on the same principle as when it results from great disease of the mitral valve : viz. a powerful obstacle to the transmission of blood out of the lungs through the left side of the heart, the obstacle here consisting in the weakness of the organ and its consequent inability to propel its contents.

CHAPTER V.

INDURATION OF THE HEART.

The muscular substance of the heart sometimes undergoes induration. Corvisart has found it carried to such an extent that the heart, when struck, sounded like a dice-box or hollow horn vessel, and the scalpel, on making an incision, experienced great resistance, and produced a singular crepitating noise. Yet the fleshy substance possessed its proper colour, and did not appear converted into either an osseous, a cartilaginous, or any similar substance. This affection is very rare. Laennec and Bertin have met with it affording a resistance to the scalpel, but not causing the crepitating noise; and the same has occurred to myself. It generally occupies the whole of a ventricle, but sometimes only a portion; and it may accompany any state of the organ as to size, though most commonly it is conjoined with hypertrophy.

It consists, I apprehend, not merely, as Laennec supposed, in an increase, but in a perversion of nutrition, being somewhat different from that firmness which the heart frequently acquires by hypertrophy. M. M. Bertin and Bouillaud, with I think the majority of authors, regard it as one of the products of chronic inflammation.

Induration, according to Laennec, increases the impulse of the heart. The firmest hearts with which he had met, were also those which gave the strongest impulse. But it is conceivable that when the induration proceeds beyond a certain point, it must, as Corvisart thought, render the contraction of the ventricles more difficult, and their movements more limited.

The treatment of induration with increased action of the heart is identical with that of hypertrophy.

CHAPTER VI.

ADIPOSE AND GREASY DEGENERATIONS OF THE HEART.

Excess of Fat.—In individuals remarkable for obesity, and occasionally in others of only moderate *embonpoint*, the heart is sometimes greatly overloaded with fat, which, deposited between the pericardium and the muscular substance, not only covers the organ externally, but frequently penetrates a considerable depth between its fibres; whilst the walls themselves, as if losing (probably by the pressure) what the adipose tissue gains, become attenuated and flabby.

The older authors imagined that this affection was the cause of more or less severe symptoms, and even of sudden death. Corvisart thinks that an enormous accumulation might sometimes produce such an effect, though, in the persons in whom he had met with very fat hearts, he had seen nothing which could prove to him "that the state was morbid, that is to say, carried to such a point as constantly to derange the function of the organ, and thus constitute a malady." The experience of Laennec has led him to the same conclusions.

Appended to the present article are three cases, which lead me to suspect that fat *does* impede the action of the heart and obstruct the circulation; and that its signs, so far as I can yet judge, are, 1. diminution of the sounds—especially the first; 2. irregular pulse, without valvular disease; 3. "oppression" or even pain in the præcordial region, with general signs of a retarded circulation, producing cerebral, hepatic, and other congestions. These signs, taken in conjunction, are peculiar; because, while No. 1 is proper to simple hypertrophy, Nos. 2 and 3 are foreign to its early stages: the aggregate therefore probably denotes an encumbrance of the organ with fat, as will appear from the subjoined cases.

It would be natural to suppose, that the substitution of adipose for muscular tissue, and the extreme attenuation which the walls, especially the apex and the posterior part of the right ventricle, sometimes undergo from this cause, would be eminently favourable to rupure of the organ; yet this accident is very rarely the result. Morgagni has seen it, but Bertin has only met with a case of rupture of the auricle, while Corvisart and Laennec have not met with an instance at all. The alteration described is different from that denominated—

Greasy Degeneration of the Heart.—This, according to Laennec, is " an infiltration of the muscular substance with a matter which presents all the physical and chemical properties of grease; it is an alteration exactly similar to the greasy degeneration which Haller and Vicq-d'Azyr have observed in the muscles. Laennec has never found it but in a very small portion of the heart, and only near the point. It was of a pale yellowish colour, like dead leaves, and therefore very similar to certain varieties of softening; but he thinks that it may be distinguished from this, by its strongly greasing paper between which it is pressed. I have seen a remarkable case in which a degeneration of this kind occupied the greater part of both ventricles.

Atrophy and œdema of the adipose tissue. The former sometimes accompanies general emaciation, and the latter presents itself in cases of universal dropsy.

Two of the subjoined cases were not attended with dissections, and the signs were therefore only presumptive. The third, presenting analogous signs, was verified by dissection.

Oppression of heart; p. irregular; impulse increased; sounds diminished; cerebral congestion; presumed fatty heart.

Case 1. P. . . . . . . d, Esq., æt. 40, a large, robust, and rather plethoric man. For two or three years, has been subject to occasional giddiness, stupor, loss of memory, numbness of the right arm, and difficult articulation, with palpitation, "oppression in the heart," and pain striking to the sternum. B. costive; dyspepsia. P. at present intermittent, and neither full nor hard, but contracted. *Impulse* of the heart considerably increased and heaving. *Sounds.* 1st. inaudible when the heart beats 70; barely audible when 90: 2d sound, feeble. No murmur.

Remarks. I do not recollect ever to have heard the 1st sound so suppressed, both on the right and left side, as in this case: yet the impulse was strong and heaving! There are only two states which can easily account for the deficiency of sound: viz. either hypertrophy with contraction, or a fatty state of the heart —which had been suggested to the patient. The event must show. I can imagine that fat, partly by encumbering the heart's action, and partly by increasing the thickness of the walls through which the sound has to be transmitted, may be capable of occasioning the suppression of the first sound.

On the 11th of March, 1836, a week after my previous examination, he had an attack of cerebral congestion, with difficult articulation, numbness and formication of the right arm, confusion of memory, pulse irregular, &c. (V. S. Enema purg.—Haust. Purg.—Capiti raso lotio frigida.—Emplast. Lyttæ occipiti.—℞ cal. gr iij, opii gr ss, 4^{tis} h.—Slop diet.) On the following day, the blood was found slightly buffed; numbness of arm gone; articulation distinct, but slow; pupils rather sluggish; no headache (contr. pil. cal. c. opio). The mouth was slightly affected by the mercury, which was diminished as the symptoms declined. At the end of three weeks, he was perfectly well of the cerebral attack. I then found the sound of the heart more audible, the impulse diminished, and the pulse regular and less contracted —symptoms indicating that the organ disgorged itself more freely.

Was ordered to avoid exercise, mental excitement, all stimulant drinks, and to live principally on fish and fowl instead of animal food, for at least two years.

During this period he lost fat, but gained strength; the sound became louder, the impulse less, and the pulse larger and more regular. Was this the result of absorption of fat, or of diminution of hypertrophy with contraction? The former is more probable. I have not seen the patient during the past year.

Angina; p. irregular; impulse increased; sounds diminished; presumed fatty heart.

Case 2. S. . . . . n, Esq., a medical practitioner, æt. 40, extremely fat and florid, (17¾ stone,) consulted me Aug. 3, 1836. Occasional slight pain in the heart, but more frequently "oppression." Little inconvenience from moderate exercise. Digestion excellent. B. regular. P. irregular, and some beats are fuller than in a healthy male of average size. (Therefore, no contraction of the left ventricle in this case.) *Impulse* slightly increased, but not easily felt, from obesity. *Sounds:* both rather dull, especially the first, and more, I think, than is accounted for by the external obesity. No murmur. He has often been cupped and leeched, which eased the oppression. Lives low.

Diagnosis. Hypertrophy is indicated by the increased impulse and the occasional large beats of the pulse. As permanent irregularity of the pulse is foreign to hypertrophy in a vigorous, healthy subject, the irregularity is probably referable to fat encumbering the organ. The same occurred in Mr. P.'s case. Investigate the subject. Important to discover whether fat produces these symptoms, because the disease may perhaps be very curable by liq. potassæ, iodine, dry diet, &c.

(Local bleeding to ℥ x or xii, now, and a fortnight hence: subsequently, every month, till contraindicated.—Lower diet: less animal food.—Liq. potass. m xx to XL ter die.—To weigh himself periodically.)

In six weeks he lost 11 lb. by the application of 100 leeches, which drew 44 ounces of blood. He had raised the doses of liq. potass. to ʒij daily. All pain and oppression had subsided, and he felt much relieved without being weaker. The p. was still irregular and intermittent, but moderately full and strong: first sound, louder—almost as distinct as natural: impulse still slightly increased when a strong beat is felt.

During the ensuing six weeks, he lost 23 ounces of blood by 56 leeches; by which his weight sustained a further reduction of 2½ lbs. He omitted the liq. potass. All the symptoms continued to improve, but the sounds were still not quite distinct enough.

During the subsequent four months, he very seldom experienced angina; and it was always relieved by leeches and vini colch. m xl, which acted in an hour as a free diuretic. The first sound became as distinct as natural, and he felt active and well. The pulse was still intermittent and irregular, but much less so than formerly. *Remark.* I imagine that the walls are thinner and less encumbered with fat, whence freer action. (Contr. omnia pro re nata). Up to the present time, March 1839, he has maintained his ground.

Oppression at the heart; p. irregular; sounds weak; hepatic congestion; fatty heart found.

Case 3. Mr. P. . . . . . . r, (whom I attended with Mr. Linnecar, Mr. Lucas, and Dr. Chambers,) æt. 50; stout, fat, (15 or 16 stone.) Occasionally, "oppression" at the heart, and pain down the inside of the left arm. Can walk up stairs and up hill with little inconvenience. P. very irregular and unequal, and a strong beat occasionally. Flatulence; b. regular from aperients. *Impulse* pretty strong when the pulse presents a strong beat. *Sounds:* both very dull. The first is, I think, but am not quite sure, attended with a murmur over the aortic valves, with the strong beats of the pulse only. Three years ago, had inflammation of the heart.

Diagnosis. Either simple hypertrophy, or, as this does not properly cause an irregular pulse, fattiness of the heart in addition. (V. S. ad ℥ vi, subinde.—Liq. potass. ʒ½ ad ʒ i ter die.—Aperients; lavements; meat and fish on alternate days.—No wine, spirits, or malt liquors, and a dry diet.—Quiet.—To lose a stone weight).

May 1, 1838. After three weeks of the treatment, the p. was fuller and less irregular; the beat of the heart stronger; the first sound rather louder. Oppression of heart and pain of arm gone; felt lighter and better.

In five weeks more, he was still better in all respects. Impulse and first sound stronger; p., though intermittent, was very full and pretty strong (hypertrophy with dilatation). No loss of weight, but abdomen diminished. He continued to improve up to the end of Oct., when he was attacked with some acute affection, for which he was treated by another physician. Six weeks later, I again attended him in consultation. The liver was now so large as to descend below the umbilicus, and he was deeply tinged with icterus. There were the usual symptoms, in a marked form, of universal venous retardation. By mercury and aperients the liver was brought almost within the margin of the ribs, but the retardation continued, and he died, greatly emaciated, in about three weeks.

Autopsy. A layer of fat upwards of half an inch thick occupied the anterior mediastinum, in front of the heart. The anterior and lower half of the right ventricle was covered with a layer of fat about a quarter of an inch thick. The heart was about one half larger than natural. The left ventricle was three quarters of an inch thick, and its cavity dilated. The right valves were sound. The left were slightly thickened by fibrous hypertrophy and steatoma, but were of natural dimensions, and also flexible and efficient. A few steatomatous depositions, with slight corrugation, existed at the origin of the aorta, and occasioned the slight murmur heard in that situation. The heart was soft and flabby. Liver, not enlarged; of the nutmeg appearance.

Remarks. As the corpse was emaciated, it is probable that, during life, the accumulation of fat had been more considerable. It is also probable that the fat had encumbered and embarrassed the organ; since it is not usual for such a degree of hypertrophy with dilatation as existed in this case, to produce irregularity of the pulse and diminution of the sounds, except in feeble, exhausted subjects—which was not originally the case in the present instance. The hepatic tumefaction was, I presume, occasioned by the venous retardation; first, because it came on suddenly; secondly, because it was speedily removed.

CHAPTER VII.

OSSEOUS, CARTILAGINOUS, AND OTHER ACCIDENTAL PRODUCTIONS CONNECTED WITH THE MUSCULAR SUBSTANCE OF THE HEART, AND WITH THE PERICARDIUM.

Osseous and cartilaginous productions penetrating into, and replacing the muscular substance, are very rare. They originate, not in the muscular fibre itself, but either in the fibrous tissue of the pericardium, or in the cellular tissue uniting it or the endocardium to the heart, and dipping in between the fasciculi of muscular fibres. This is in accordance with the general laws of embryogony and of the animal scale: namely, that certain tissues only are convertible into certain others; that cellular tissue is the matrix, as it were, of all others, and that cellular may be transformed into fibrous, fibrous into cartilaginous, and cartilaginous into osseous.

Corvisart has seen the point of the heart, in its whole thickness, and the left columnæ carneæ, converted into cartilage. I have seen the same at the base. Fig. 20 is an ossified aneurism. Burns has seen the ventricles perfectly ossified, so as to resemble the bones of the cranium. Haller, Filling and Bertin have seen partial ossifications. M. Renauldin has found the left ventricle converted into a real petrifaction, which had a sandy appearance in some parts, and in others resembled a saline crystallization. In all these cases, the disappearance of the muscular fibre is referable to atrophy, resulting from compression by the encroachment of the new productions.

Cartilaginous incrustations occasionally exist between the lining membrane and the muscular substance. Kreysig found one in an ossified state. Mr. Thurnam describes several cases of fibrous, cartilaginous, and osseous disease of the muscular substance, in connexion with real aneurism of the left ventricle (see p. 322).

All these transformations are generally results of pericarditis or endocarditis.

Laennec feels persuaded that an osseous or cartilaginous induration of a large portion of the heart, as a whole ventricle or half the organ, could be recognised with the cylinder, by a very marked augmentation, and some particular modifications, of the sound of the organ. He thinks that cases of this nature are amongst those in which the sound of the heart can be heard at a certain distance from the patient. These anticipations have not been realized; and it is now very apparent why they should not. For, as it has been shown in the experiments at p. 25 et seq. that the first sound of the heart is occasioned by the extension of the muscular walls and of the auricular valves, it is obvious that, when the force of this extension is diminished by the substitution of cartilage or bone for muscular fibre, the sound must sustain a corresponding diminution of intensity. Accordingly, in some of Mr. Thurnam's cases, the sound actually was enfeebled. The only case in which I can imagine it augmented, would be, when the apex is indurated and creates an adventitious sound or metallic cliquetis, by impinging against the inferior margin of the fifth rib, in the manner explained at p. 41.

It is probable that fibrous, cartilaginous, and osseous transformations of the muscular substance would generally be attended with a murmur, because they almost always implicate the valves. If the surfaces of the pericardium were roughened by the disease, an attrition-murmur would be the result. On this subject, as on real aneurisms of the left ventricle, a new series of observations is required; the physical signs, in the cases that have hitherto occurred, having been very imperfectly explored.

Osseous and cartilaginous depositions sometimes take place in the reflected pericardium, being originally seated either in the subserous cellular tissue, or in the fibrous layer itself. Though they do not properly fall amongst the diseases of the muscular substance, they are introduced here, because they are not of sufficient importance to form a separate chapter. Laennec met with an osseous deposition between the fibrous and serous layers, which formed a band from one to two fingers broad, completely encircling the heart, and sending off triangular processes towards the apex. (De l'Auscult. tom. ii. p. 675). In other instances, in which the concretion has formed a similar ring, or a case nearly enclosing the whole organ, it has sometimes given off processes which penetrated the muscular substance, (Latham, Lond. Med. Gaz. vol. iii. p. 7,) and reached even into the cavities. The general symptoms have been those of great obstruction of the circulation. Dr. Elliotson relates two cases in which masses of cartilage connected with the pericardium compressed the pulmonary artery, and created a murmur.

As osseous or cartilaginous degeneration of the heart and pericardium is incurable, the treatment can only be palliative.

Tubercles (see case of a *Genevese*) and tumours of a carcinomatous nature have been found in the substance of the heart. Recamier has seen the organ converted in part into scirrhous matter like the skin of bacon, in a subject who had also carcinomatous tumours in the lungs. M. M. Laennec, Andral, Bayle, Bouillaud, and others, have found cancer in the heart. The total number of cases recorded amounts to about a dozen.

From these it appears that, in the heart, as in other organs, carcinomatous productions, both scirrhous and encephaloid, may be developed in two principal forms, that of *isolated tumours*, and that of *interstitial infiltration*. They rarely exist without similar productions in other organs, especially the lungs. There can be no doubt that cancer, if sufficiently extensive, would impede the action of the heart and obstruct the circulation; but the cases on record are too few to afford data for a general history of the disease. Cruveilhier has delineated melanosis forming numerous tumours under the pericardium and in the substance of the heart. I have seen similar cases. Serous cysts and vesicular worms, (apparently the *cysticercus finnus* of Rudolphi,) have also been found in the heart.

CHAPTER VIII.

ATROPHY OF THE HEART.

Atrophy consists in deficient nutrition, and the heart, like any other muscle, is liable to it. The heart of an adult was found by Burns not larger than that of a new-born infant, and the heart of a female of twenty-six not larger than that of a child of six. Bertin gives a similar case (66): the writer has met with the same; and numerous other instances are on record.

Atrophy generally takes place under the influence of those causes which produce general emaciation: chronic diseases, for instance; as phthisis, diabetes, chronic dysentery, cancer and malignant affections in general. Excessive bleeding is another cause. Laennec adduces an instance resulting from the treatment of Albertini and Valsalva employed to cure hypertrophy. Finally, protracted compression by fluid effused within the pericardium, as in cases of chronic pericarditis, may produce the effect, and Bouillaud relates cases in which the same resulted from compression by "enormous masses of false membrane" (Traité, i. 448).

The heart, when atrophous, generally contracts upon itself, so as to diminish its cavities, while its walls do not become materially thinner, and sometimes become even thicker than natural. In the latter case, the affection must not be mistaken for hypertrophy, and the error may be avoided not only by remarking the general diminution of the volume of the heart, but also the shrivelled and wrinkled appearance of its exterior.

Atrophy may also co-exist with dilatation, namely, when the walls are so thin that the total volume of the muscular substance is diminished.

Diminution of the volume of the heart does not appear to produce symptoms which entitle it to be ranked as a disease. Individuals who present this peculiarity are perhaps less subject to inflammatory complaints than others, though they are more prone to anæmia, to fainting from slight causes, and to nervous affections. It is remarkable that women, who are more subject to these ailments than men, have in general smaller hearts.

The treatment for atrophy is principally that of its causes: otherwise, it is the same as that for dilatation.

CHAPTER IX.

DISEASES OF THE VALVES AND ORIFICES OF THE HEART.

SECTION I.

ANATOMICAL CHARACTERS, WITH PREDISPOSING AND EXCITING CAUSES, OF DISEASES OF THE VALVES.

We resume this subject at the point where we left it at the end of the section on the anatomical characters of chronic endocarditis, p. 212.

The valves and chordæ tendineæ consist, according to the best authorities, of fibrous tissue interposed between a production and reduplication of the lining membrane of the heart. The fibrous tissue is prolonged from a dense, whitish zone of the same, which encircles each of the orifices of the heart, and is, as it were, the tendon or point of attachment into which the muscular fibres of the organ are inserted. The lining membrane of the heart, according to Bichât, approximates closely in character to serous membranes: the valves, therefore, may be said to consist of *fibro-serous* tissue. Now, the fibrous tissue in general is remarkable for its proneness to cartilaginous and osseous degeneration; whence we derive an explanation of the fact, that the valves and orifices of the heart are frequently affected with these degenerations, while the cavities, where they are invested solely by the lining membrane, are in a great measure exempt. Though disease occupy a valve universally, it generally stops abruptly where the serous membrane is continued from the circular zone, or the extremities of the chordæ tendineæ, upon the muscular substance. In a few instances it advances farther; but I have never seen it attack the membrane of the muscular substance without being connected with, and apparently propagated from, disease of the valves: and, in these cases, a conversion of subserous cellular tissue into cellulo-fibrous had preceded the transformation into cartilage or bone.*

It appears, then, that the disease is dependent for its origin on the fibrous and not on the serous tissue; in corroboration of which view, it may be stated that, where the fibrous tissue is most abundant,—namely, at the base and the free margin of the valves, cartilaginous and osseous depositions are the most frequent and extensive: and again, it is common to find the valves encumbered with large masses of cartilage from which the internal membrane can be peeled off in its natural thin and transparent state. In these cases the surface of the morbid deposition is smooth and equable; and it is seldom until it becomes corrugated, rugged and knotty, that the internal membrane is implicated in the disease. Calcareous depositions, in the same way, seem always to commence underneath the membrane. In a case under my observation, (Fig. 15,) in which two rings of bone as thick as writing quills encircled the left orifices of the heart respectively, the membrane was stretched like a blue film over the whole of the aortic, and the greater part of the mitral ring.

Valvular disease is much more rare on the right, than on the left side of the heart. Bichât, indeed, denied its existence at all in the former situation, but his opinion has been fully disproved. Morgagni, Vieussens, Hunauld, Horn, Cruwel, Corvisart, Burns, Bertin, Louis, Laennec, Bouillaud, Latham, Clendinning, and many others, have all met with instances of disease of the right valves. Dr. Latham thinks that in one-third of the cases in which he has seen disease of the left valves, it has existed in the right also. Up to the year 1831, I had notes of eight cases in which it existed in the right, and I could recollect several others. In six of the eight the left side was simultaneously affected, and generally to a much greater extent; but the proportion which the whole number mentioned bore to the cases that I had seen of disease on the left side, was less than that indicated by Dr. Latham, not exceeding, I think, one in four and a half to five. Since 1831, I have reason to believe, from the examination of a vast number of cases of valvular disease, mostly without, but occasionally with dissection, that the proportion of affections on the right side, as compared with the left, is very much smaller than I have specified above. I cannot state, numerically, the exact proportion, as I have not leisure at present to analyse 10,000 cases, which I calculate to yield about four per cent., or 400 cases, of valvular disease; but my general impression is, that, out of the 400, I have not, at the utmost, met with 20 cases of disease of the right valves,—which would only be five per cent., or 1 in 20. Dr. Clendinning has met with about 1 in 16, out of 100 cases, as exhibited in the following statement, with which he has obligingly favoured me:

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* These present the most familiar instances of *Analogous Transformations*, by which term is meant a conversion of one tissue into some other *natural* to the system, in contradistinction to cancer, tubercle and others, which present *no analogy* to anything in the healthy system, and are therefore called *non-analogous* productions.

But though one tissue may be transformed into another natural to the system, it cannot be transformed into *any* other, but only into *certain* others. The laws which regulate the selection are exceedingly curious, interesting, and instructive; for they are exactly those which preside over the growth of the human embryo, or are exhibited in the "series of animals." The young reader is strongly recommended to make himself well acquainted with them (see Andral's Path. Anat. vol. i. chap. 4). Here, it is sufficient to say that no transformations are more common than those of cellular tissue into fibrous, fibrous into cartilaginous, and cartilaginous into osseous. "These aberrations from the natural nutrition of the part," says Andral, "are preceded in many cases by irritation, (inflammation,) but *neither constantly, nor necessarily so*......and the knowledge which we now possess on the laws of embryogony, as well as of those which regulate the nutrition of different animals, enables us to conceive how every species of transformation of tissue may occur independently of any antecedent irritation" (Ibid. p. 292). The reader is referred back to *chronic arteritis*, p. 227, for an account of the circumstances under which valvular and arterial diseases may not have originated in inflammation.

Valves of the left side *alone*,	92 ·6,	or $\frac{9}{10}$ths,
. . . . . right side *alone*,	2 ·1,	or $\frac{1}{47}$th,
. . . . . both sides,	6 ·31,	or $\frac{1}{16}$th,

He adds, however, that he neglected to record some instances which "might fairly be presumed to have occurred mainly, if not exclusively, under the first head." This would reduce his proportion below $\frac{1}{16}$th, and bring it nearer to mine, namely $\frac{1}{20}$th. I suspect, however, that it will eventually prove to be lower still. It is remarkable that in all my own cases, except Lady R., and nearly all those of the authors quoted, (with the exception of Dr. Latham, who is silent on this point, and Dr. Clendinning, whom I have not had the opportunity of consulting,) the induration on the right side was merely fibrous or cartilaginous, and never osseous. When the two sides are affected at once, it very rarely happens that the disease on the right is greater than that on the left; in general it is much less, being comparatively slight or incipient.

Respecting the cause of the remarkable difference which the two sides of the heart exhibit in their liability to induration, authors have not been agreed. Corvisart attributed it to a more decidedly fibrous organisation of the left valves, in virtue of which they are "more disposed to receive the matter that is to transform them into cartilage, or the calcareous salts that impart to them an osseous or stony hardness." M. M. Bertin and Bouillaud have ascribed the difference to the different nature of the blood that traverses the two sides respectively, the left receiving blood of a more vital, more stimulating, more irritating quality than that by which the right cavities are moistened. Laennec does not offer a decisive opinion.

Without pretending to decide whether the latter cause conspires, or not, to produce the effect, I entertain no doubt that the opinion of Corvisart is substantially correct; for I have already repeatedly shown that it is principally the fibrous tissue which undergoes transformation into cartilage and bone, both under the influence of inflammation and independent of it. It also happens that both these classes of exciting causes, the inflammatory and uninflammatory, are most in operation on the left side of the heart; for it is here that endocarditis is of most frequent occurrence, and that the valves are most strained by the greater power of the left ventricle and the stronger retrograde pressure of the aortic blood: and it has been shown at p. 229, that inordinate straining of the valves is a cause of their hypertrophy and transformation into cartilage and bone.

The appearances of valvular induration are somewhat different, according as the disease occupies the auriculo-ventricular, or the arterial valves; the cause of which is to be found in the difference which naturally subsists between the valves themselves. I shall therefore describe the degenerations of the two classes of valves separately. It may be premised that there is no essential difference but in degree and frequency of occurrence, between the degenerations on the two sides of the heart; consequently, a description drawn from the left will apply to the right.

Induration of the Mitral Valve.—The appearance presented by the indurated mitral valve differs according as the disease occupies the base, the margin, or the whole of the valve.

When the whole is affected with fibro-cartilaginous degeneration, the valve is generally contracted throughout, and what is lost in space appears, as it were, expended in thickening the free border; for this is converted either into a ring, an oval-shaped collar, or a transverse slit like a button-hole (Figs. 5, 7, 12). The size of the aperture is various. I have seen it of all sizes from an inch to a quarter of an inch in its longest diameter. The thickness of the border likewise varies. I have seen it equal a writing quill. When the valve is thus contracted, it generally projects more or less, in a funnel shape, into the cavity of the ventricle. In one case I found it project so far that the columnæ carneæ were inserted immediately into the ring, the chordæ tendineæ having disappeared. The surface of the induration is smooth, polished, and translucent until the disease throws out osseous or other excrescences, which, interfering with the integrity of the investing membrane, render it corrugated, rugged, and opake. Before ossification takes place, the induration described sometimes presents a truly cartilaginous hardness, and sometimes the consistence of fibro-cartilage, or only that of fibrous tissue. When divided, the aspect of the section varies according as the disease is cartilaginous, fibro-cartilaginous, or fibrous.

In a more advanced degree, cartilaginous induration is transformed into imperfect bone. It seldom happens, however, that more than a very small proportion of the cartilaginous mass is ossified, and the change takes place sometimes at its surface, and sometimes deep in its substance. The bone produced does not exhibit the fibrous structure and peculiar arrangement of natural bone; though, as it contains a large proportion of cartilage, it may be presumed to possess more or less vascularity and vitality.

There is another species of osseous induration of the valves, which is essentially different from the above, inasmuch as it consists of calcareous matter in great predominance, and, like vesical calculi, has no vitality. It presents itself under the form of small, polished, and semi-transparent scales; or of minute, yellowish, opake granules, the agglomeration of which forms concretions of various dimensions, from a mere point to the size of a horse-bean.

The deposition commences underneath the lining membrane, and generally in a small patch of indurated, cheese-like *steatomatous* matter; the surrounding parts being healthy. The scales lie flat and superficial under the membrane, while the granules penetrate more or less deeply into the subjacent tissues. When either the scales or the granules enlarge, and their surfaces become rugged or acuminated, they cause absorption of the internal membrane, and come in immediate contact with the blood.

Some authors believe that ossifications of this description are *natural* to old people, because they occur in the majority of those who have attained the age of sixty. Whatever be the character of the ossification, whether it be mixed with cartilage or purely calcareous, to me it appears to be a morbid production. The circumstance of its occurring in the majority of persons above the age of sixty, does not militate against this view: for, as the elasticity of the arterial, as of all the other tissues, is diminished by age, the valves of the heart and the coats of the arteries are, in the aged, less capable of resisting the distending force of the blood, and are therefore more liable to disease. Nor does the circumstance of the ossification being more calcareous and less cartilaginous in the old than in the young prove that, in the former, it is a *natural* change. It confirms, indeed, what is proved by every part of the bony tissue; viz. that in age the ossific tendency is greater; but it does not, for this reason, follow that the tendency is natural when it displays itself in an unnatural situation, as in the heart and arteries. I find this opinion expressed in almost the same words by Andral. "The process of ossification naturally increases in extent as the individual advances in life: but, notwithstanding the general physiological nature of this process, it may constitute a true pathological condition, by interfering with the due accomplishment of vital function, as in certain cases of ossification of the heart and arteries" (Path. Anat. i. 368).

Sometimes the membranous portion and free margin of the valve are healthy, while the fibrous zone at the base is cartilaginous, or beset with small calcareous incrustations, or, as sometimes happens, its whole substance is converted into a thick ring of bone (Fig. 15). By these depositions at the base of the valve, the orifice is more or less contracted, while the valve itself may remain capable of closing. In many cases, again, the base and middle are sound, and the free margin alone is diseased, its conical processes forming adhesions with each other, and contracting the circumference of the valve to such an extent as almost completely to close the orifice (Mrs. —l—n). It is not uncommon to find the margin studded with vegetations, small cartilaginous nodules, or roundish calcareous granules, which prevent the accurate adaptation of the edges to each other, and allow regurgitation during the ventricular contraction. Sometimes, the only diseased appearance that the valve presents, consists in brittle scales or patches of pure phosphate of lime between the two component layers of the membranous portion, which they occasionally rupture, and thus come in immediate contact with the blood. Sometimes, again, the only material lesion of a valve is shortening and thickening of the tendinous chords, which prevent the valve from completely closing during the ventricular systole (Figs. 5, 7, and 12). This was unknown as an important lesion till it was pointed out in the first edition of this work; and, even up to the present day, I see it perpetually overlooked in the dissecting-room by those whose attention has never been specifically directed to it: yet, from being attended with regurgitation, it constitutes one of the worst varieties of disease of the valves.

Sometimes, though rarely, the same regurgitation is occasioned by one of the membranous expansions of an auricular valve having adhered, by inflammation, to the walls of the ventricle; and it is principally the posterior layer that becomes thus adherent, because it is less moveable.*

Another affection of the valves, whether auricular or semilunar, occasioning regurgitation, is atrophy. By this, I have seen the membranous expansions of the mitral valve reduced to a mere reticulated web, and the aortic valves perforated in five or six places. The affection commonly occurs in connexion with general atrophy and anæmia. It has been fully described by Dr. Kingston (Medico-Chirurg. Trans.).

I may here add, that, without any disease whatever of the valve itself, regurgitation may take place when, in consequence of dilatation of the auricular orifice, the valve is not large enough to close it—a condition of parts which I have occasionally met with in cases of great dilatation of the left ventricle.

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* M. Bouillaud is mistaken in supposing himself the discoverer of this adhesion (See his Traité, ii. 188). Dr. Elliotson described it five years previously in his Lumleyan Lectures.

Induraiion of the Aortic Valves.—Induration of the aortic valves, like that of the mitral, is more frequent and extensive at the base and free border, than in the intermediate space. At the border, it originates more especially in the corpora sesamoidea, because they contain more fibrous tissue: hence these bodies are sometimes enlarged by cartilage to the size of peas. I have seen the margin contracted by fibro-cartilage into a ring a quarter of an inch in diameter. (Hedgley, Fig. 17). I have seen the margin of the individual valves thickened and contracted, so that the were too small to close the orifice (See Figs. 6 and 14). I have seen the corners of the valves adhere, from inflammation, to the arterial walls, so as to leave an interval between each two valves, which permitted regurgitation (Fig. 11, *b*.). I have seen a similar interval occasioned by an aneurism of the aorta stretching the orgin of the pulmonary artery where the affected valves were seated (Fig. 12, *b*). The valves are sometimes thickened, nodulated and corrugated by an opake yellow degeneration, consisting of a mixture of cartilaginous and steatomatous matter. I have repeatedly seen the angles of the valves detached from their bases and partially wasted away by this deganeration; so that, adhering by their centres only, they hung loose into the artery, and were destitute of fulcra by which to oppose the reflux of blood from the aorta (Copas). In Fig. 14, A, they hung loose into the ventricle, and two valves were together. In another instance, the same disease had undermined and more or less detached the bases of all the valves throughout nearly their whole length; and, under one of them, it had led to the formation of a canal, as wide as the little finger, beneath the lining membrane of the heart, leading to an aneurism in the muscular substance of the septum between the left auricle and ventricle (Case of Brown). The same is seen in Fig. 20. I have once seen the edge of a valve rent, so that a flap hung back and allowed regurgitation (Fig. 11, *a*). The same valve contained a perforation (Fig. 10, *a*).

Such are the cartilaginous and steatomatous degenerations of the aortic valves. The osseous, of which we have next to speak, are perhaps as frequent in the aortic as in the mitral valves. The ossification may be either pure, or combined with cartilage.

In one case under my observation, an irregular, scabrous, and denuded concretion, the size of a pea, occupied the edge of one of the valves, and projected into the cavity of the artery. (Porter, Fig. 16). In another case, a similar mass, of a conical shape, sprang from the base between two of the valves, and presented its apex towards the centre of the vessel. (May). Smaller concretions of this description, and in this position, are common. M. Bertin saw an ossification of one of the aortic valves which had attained the size of a pigeon's egg (Obs. 53). In one of my cases, already alluded to, the fibrous zone encircling the base of the aortic orifice was converted into a ring of bone as thick as a quill (Fig. 15).

When the ossification is confined to the margin and base, while the middle portion is still healthy over a certain extent, the valve, if its thickening is not very considerable, may still rise and fall, and not offer any marked obstacle to the circulation. But when the ossification pervades the middle portion of the valves, they shrink, become soldered together, or curl up upon themselves, in the direction either of their concavity or convexity, so as to present a rude representation of certain sea-shells. In this state they may become immovable. If curled forwards, they remain applied along the walls of the aorta, and oppose no other impediment to the course of the blood than what results from the thickness of the ossification. They then permit regurgitation. If curled backwards, they remain fixed in the fallen or shut position, and considerably contract the orifice, as well as permit regurgitation (Fig. 18). Not unfrequently, one of the three valves is curled in an opposite direction to the other two. Corvisart has seen all three ossified in the closed position, and they would only have left an extremely narrow cleft for the passage of the blood, had not one retained sufficient mobility at its base to perform a movement which augmented, by a line or two, the width of the cleft.

Induration of the Valves at the right side of the Heart.—Induration of the right or venous valves is, as already stated, almost always simply cartilaginous or fibro-cartilaginous, (Fig. 12,) and is comparatively rare, not existing in perhaps more than about one case in sixteen, twenty, or more, of disease in the left valves. It seldom presents itself without being accompanied by disease of the left valves also, and it is, in general, less advanced than the latter (Anderson, Sharpe). The tricuspid is more frequently affected than the pulmonic valves. I have never seen the latter diseased, but I have once found them incapable of closing the orifice in consequence of dilatation of the artery, (Weatherly,) and I have seen the orifice contracted to the diameter of a quill, an inch below the valves (Collins). M. Bertin has seen the valves themselves contracted into a circular aperture only two lines and a half in diameter. A few other lesions foreign to the valves themselves, have been described at p. 75. As already stated, disease of the right valves, whether cartilaginous or osseous, only differs from that of the left in frequency and extent, its characters being essentially the same.

Predisposing Causes of Valvular Disease.—These are, the larger proportion of fibrous tissue in the valves of the left side; advanced age, a cachectic state of the system from inebriety, mercurio-syphilitic disease, gout, hard labour, insufficient food, &c.

Exciting Causes.—1. Inflammation of the internal membrane of the heart, generally connected with acute rheumatism. This is the most frequent and important cause. It is fully noticed under the anatomical characters of endocarditis, pp. 208 and 211.

2. Such causes as overstrain the valves by increasing the force of the circulation; namely, violent and long-continued corporeal efforts, hypertrophy with dilatation, protracted nervous palpitation. These causes occasion hypertrophy of the fibrous tissue of the valves, which may subsequently pass into cartilaginous and osseous disease. They are more fully considered at p. 227. In a few cases, I have known violent efforts occasion laceration of a valve, and the injury has induced endocarditis. The immediate symptoms are noticed at p. 198.*

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* Other cases of ruptured valves have been described by Adams, Cheyne, and Townsend. On rupture of the heart itself, see Dict. de Med. Cœur, Rupture, by Ollivier.

SECTION II.

ANATOMICAL CHARACTERS AND CAUSES OF WARTY VEGETATIONS OF THE VALVES.

THESE excrescences bear a close resemblance to venereal warty vegetations on the external organs of generation. Their form is in general irregularly spherical, oval, or cylindrical: their size varies between that of a small pin's head and a large pea, but when isolated they are occasionally as large as a horse-bean. Their surface is polished, but often lobulated like a raspberry: they are found either isolated, in clusters, or in closely agglomerated patches like cauliflowers. Their number is various: sometimes there are only one or two, and sometimes they pervade the whole of the valves, the tendinous cords and a great portion of the auricle (Dolan). Their colour, occasionally of a greyish or yellowish white, is more commonly heightened, universally or in parts, with pink or red of greater or less depth. Their texture is fleshy and slightly translucent, like the exuberant granulations of an ulcer. Their consistence is variable; in general they are soft and humid, as if only recently and imperfectly organized; and they can then be easily scraped off with the handle of the scalpel; but sometimes they are firm, like fibro-cartilage, creak under the knife, and cannot be detached without tearing with the nail, or cutting with the edge of the scalpel. Firm vegetations are generally larger and more truly warty than soft.

The internal membrane of the part from which vegetations spring, is almost invariably more or less diseased. It is thickened, steatomatous or cartilaginous, ossified, ulcerated or ruptured. When vegetations grow from a diseased, but *unbroken* surface, they may be numerous, and occur in several parts at once; but when they grow from a ruptured or ulcerated edge, they are few in number, often not exceeding one or two, are generally confined to that edge exclusively, and attain a larger size than any others. I have seen them exceed a horse-bean, and with a neck two, three, or four lines long. It cannot be doubted that their origin is connected with the broken state of the membrane.

The base and free margin of the valves appear to be peculiarly favourable to the growth of warty vegetations. Along these parts, but especially the latter, they are often arranged in a single row. They occur on both sides of the heart, but less frequently on the right. The aortic and mitral valves are the parts most subject to them. They are more rare in the auricles than on the valves, especially in the right auricle. I have, however, seen one-third of the left auricle completely covered with them. (Dolan). When situated at the base, or the free margin of a valve, they encumber its movements, prevent its closure, and contract its aperture according to their size and number.

Laennec thought it "indubitable that vegetations were nothing more than small polypous or fibrinous concretions, which, being formed on the sides of the valves or auricles, become organised by a process of absorption or nutrition analogous to that which converts albuminous false membranes into adventitious membranes or cellular tissue." This opinion is unsatisfactory; for, as polypi are most common in the right cavities of the heart, vegetations ought to be so likewise,—the reverse of which is the fact. The valves, moreover, being perpetually in motion, would be the last parts to which albuminous concretions would adhere, as it is a stagnant state of the blood which is most favourable to their formation; yet the valves are the parts most subject to them. We most commonly find *real* sanguineous concretions, when of *small* size, amidst the intricacies of the columnæ carneæ, where the blood is more stagnant than elsewhere. Finally, if vegetations were merely fibrinous concretions, instead of being rare, they ought to be frequent; for, as the circumstances which, on this view, lead to their formation, are common to all persons labouring under an obstructed circulation, all, or, to say the least, many, should be affected with them. These considerations, then, render it highly improbable that vegetations are formed by mere coagulation of the blood under *ordinary* circumstances.

Kreysig attributes their formation to inflammation. M. M. Bertin and Bouillaud have espoused the same opinion, resting on the fact that vegetations bear a close analogy to the albuminous granulations occasionally found on serous membranes affected with chronic inflammation. The small and soft vegetations certainly bear this analogy—a fact of which I have satisfied myself by comparing the two as occurring in the same subject. I have also produced these vegetations in the space of an hour, by lacerating the pulmonic valves and interior of the right ventricle with the point of a hook, in an ass poisoned with woorara; (see *Autopsy*, p. 38;) whence I think it highly probable that they were occasioned by an exudation of coagulable lymph; for we know that it may exude from the surface of a cutaneous abrasion or cut within the brief period specified. The inflammatory origin of vegetations, moreover, is countenanced by the fact that the internal membrane of the part from which they spring, is almost invariably more or less thickened, steatomatous, cartilaginous, ossified, or ulcerated—lesions which *most frequently*, though not always, result from inflammation: and, further, since the signs of endocarditis have been well understood, it has occurred to myself and others to find that vegetations have generally been preceded by some tolerably distinct attack of that inflammation, usually in connexion with a rheumatic fever.* There are probably two modes in which inflammation has the effect of producing the vegetations: 1. by effusing coagulable lymph, which becomes organised, precisely as we see globular granulations produced on the pleura, pericardium, or peritoneum; 2. by imparting to the blood in contact with the inflamed part a morbid tendency to coagulate—a tendency which may be legitimately inferred to exist here, because we know, from positive observation, that it exists in local inflammations of veins and arteries, and because the fatal cases of acute endocarditis related by M. Bouillaud have actually shown that, in this affection, the blood frequently coagulates before death, and forms colourless, adherent polypi. Now, admitting that its fibrine has this morbid tendency to coagulate, it is very conceivable, as M. Bouillaud suggests, that it may be deposited on the tendinous chords and edges of the valves, agitated by alternate movements, just as we see it deposited on the rods with which we beat blood. Once deposited, it naturally becomes organised.

With respect to the large, dense, and more properly wart-like vegetations, it is consistent with analogy to suppose that their nutrition has undergone some of the capricious modifications or perversions, which we so frequently witness in chronic inflammation, and which may have caused their transformation into a dense, cellulo-fibrous tissue. Possibly, the greater friction and agitation to which large vegetations are subjected, may be the source of their altered nutrition.

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* See the remarkable case of Fenn.

The resemblance which the firmer valvular vegetations bear to venereal warts, led Corvisart to think that they might have the same venereal origin. This opinion, however, is not tenable; as extensive observation in venereal hospitals has proved that vegetations of the heart are not more common in persons affected with this disease than in others; and it is certain that they have occurred in numbers who had never been in the least degree tainted with the disease.

SECTION III.

PATHOLOGICAL EFFECTS OF DISEASE OF THE VALVES, AND MODE OF THEIR PRODUCTION.

DISEASES of the valves, whatever be their nature, whether osseous, cartilaginous, or warty, have for their common effect, to obstruct the orifices of the heart; and this they do, either by contracting the apertures, or by encumbering the valves in such a manner as to prevent them from opening and closing with suitable accuracy and facility; whence there results, either an impediment to the direct flow of the blood through the aperture, a regurgitation, or both. A mechanical obstacle is thus presented to the circulation, and, from the obstruction and embarrassment which it occasions, are derived the symptoms of valvular disease.

The general symptoms, however, when of an aggravated nature, are seldom dependent on the valvular obstruction exclusively; they are partly attributable to a co-existent disease of the muscular apparatus of the heart. For, so long as the organ remains free from dilatation, hypertrophy, or softening, the valvular disease, according to my observation, is not in general productive of great inconvenience.*

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* This opinion is strongly opposed to the favourite doctrine of M. M. Bertin and Bouillaud, and of M. Bouillaud in his later work: namely, that the symptoms of a retarded circulation are, under all circumstances, the result of a *mechanical obstacle* to the course of the blood, as a contracted valve, aortic aneurism, &c. The errors of

This opinion is founded on the following grounds. I have seen individuals, who were affected in an eminent degree with disease of the valves or of the aorta, maintain for years a very tolerable state of health so long as there was no hypertrophy or dilatation of the heart: but, in proportion as these supervened, the symptoms of valvular obstruction became more and more developed, and eventually assumed their most aggravated form.

I have reason to believe that, in these cases, the symptoms were attributable in a great measure to the hypertrophy or dilatation, because I have seen a greater valvular contraction produce less severe symptoms when the hypertrophy or dilatation was less considerable. It might be supposed that a great degree of contraction would *of itself* suffice to produce the symptoms of an obstructed circulation in their most aggravated form. This is highly probable, but it does not easily admit of demonstrative proof, as a great degree of contraction is perhaps never found, on dissection, without hypertrophy or dilatation. I therefore infer that these affections ensue as consequences of valvular contraction, and I believe, for the reasons above assigned, that they play an important part in the production of the symptoms.

It is of immense practical importance to keep in view the facts stated, namely, that valvular disease does not produce formidable symptoms until it has given rise to hypertrophy or dilatation; and that it invariably leads to these affections, unless the circulation is kept tranquil. We thus know that the most efficacious treatment of valvular disease consists in employing such prophylactic measures as are calculated to prevent the supervention of hypertrophy or dilatation, the latter usually with softening; and employing them with the same uncompromising strictness before those affections have appeared, as if they actually existed.

It remains to be explained how dilatation and hypertrophy aggravate the symptoms of valvular obstruction. I have shown

this doctrine, and the inconsistency of M. Bouillaud in maintaining it, have been pointed out at p. 252, and 302, note. I have had the satisfaction of seeing the opposite opinion in the text, come into pretty general favour in this country. One of the latest writers is Dr. Clendinning. After examining a great number of cases in the St. Marylebone Infirmary, he writes to me, in reference to 100 cases of valvular disease, that he has "come to the conclusion, whether erroneous or not, that the paramount element in cardiac pathology is muscular hypertrophy."

(see Dilatation, p. 300) that dilatation of the heart, by enfeebling the contractile power of the organ, constitutes as truly an impediment to the circulation, as a more direct mechanical obstacle. When, therefore, dilatation exists in addition to such mechanical obstacle, it is clear that the symptoms, having a twofold cause, must be doubly severe.

Hypertrophy aggravates the symptoms of valvular obstruction, because the heart, being morbidly irritable, struggles against the obstacle and falls into fits of palpitation; and as, during these, a greater quantity of blood than natural has to be transmitted through the contracted aperture, or is driven retrograde with augmented violence, the circulation is performed with increased difficulty.

It is in consequence of these reciprocal reactions of the valvular and the muscular apparatus on each other, that cases thus complicated are more severe than any others; and that capillary embarrassment, with dropsy, &c., supervenes at an earlier period, and attain a greater degree.

From what has been said here and in the parts referred to in the preceding note, the reader will judge how totally M. M. Bertin and Bouillaud, and more recently M. Bouillaud, have been wrong in referring the obstruction of the circulation to the valvular contraction exclusively, without allowing that hypertrophy, and scarcely that dilatation, contributed in any degree to the effect. Such a doctrine is not only erroneous, but dangerous, as it leads to pernicious practice. For, imagining the valvular contraction to be the only formidable part of the complaint, to it alone those authors direct their attention; and, acting on the inaccurate presumption that it is, in almost all cases, caused by, and accompanied with, inflammation, they attack it with bloodletting, general and local, abstinence, digitalis, &c.,—means which cannot remove valvular disease when once established, and which are, therefore, a useless expenditure of the patient's strength. It is true, indeed, that measures calculated to diminish the force of the circulation are useful in obviating the supervention of hypertrophy or dilatation—the paramount source of danger in these cases; but measures employed for this purpose, and which must be continued for an indefinite length of time, cannot be practised with the same activity as for the purpose of curing an inflammation. I would not be understood by this to mean, that valvular disease is *never* accompanied by inflammation, and that, when so accompanied, it should not be treated by antiphlogistic measures: but I mean that they should not be employed unless there is reasonable evidence of inflammation,—a subject which has already been fully considered under the head of chronic endocarditis (see p. 218).

SECTION IV.

SIGNS, DIAGNOSIS, PROGNOSIS, AND TERMINATIONS OF DISEASE OF THE VALVES.

General Signs of Disease of the Valves.—Whether the disease be fibrous, cartilaginous, osseous, or consist of vegetations, the general symptoms are the same, if the degree of contraction or regurgitation be equal. Keeping in view the principles developed in the preceding section, which ought to be read in connexion with the present, I should assign to disease of the valves, as its general symptoms, 1. a greatly aggravated form of the same as have already been assigned to dilatation of the ventricles; 2. certain peculiar and distinctive signs, which I shall presently describe.

1. Briefly to recapitulate these symptoms—they are, cough, copious watery expectoration in many cases, dyspnœa, orthopnœa, frightful dreams and starting from sleep, œdema of the lungs, pulmonary congestion and apoplexy, passive hæmoptysis, (i. e. sputa stained with dark or grumous blood, which occurs especially in great contraction of, or regurgitation through, the mitral valve,) turgescence of the jugular veins, lividity of the face, anasarca and dropsies in general, which in this form of disease attain their utmost degree; injection of any or all the mucous membranes; passive hæmorrhages from the same membranes; engorgement of the liver, spleen, &c., and congestion of the brain with symptoms of oppression, sometimes amounting to apoplexy; occasionally, cerebral hæmorrhage.

The reader will understand that this is an enumeration of all the worst symptoms of an advanced case. In the early stages the hæmorrhages and dropsies are generally absent, and the congestive symptoms are less marked.

When the left valves are obstructed, or permanently open, the pulmonary symptoms of the above category result from engorgement of the pulmonary vessels: when the obstruction or patescence is in the right valves, they result partly from engorgement of the bronchial veins, and partly from the quantity of blood transmitted into the lungs not being adequate to their demand, whence there is insufficient oxygenization, and its consequence, dyspnœa. In the latter case, hæmoptysis is more rare.

The symptoms affecting the system in general result from retardation of the blood in the venous system.

2. The peculiar and distinctive signs of valvular disease are the following.

a. When the disease is combined with hypertrophy or dilatation, as is almost invariably the case sooner or later, the symptoms are more severe than those of an equal degree of hypertrophy or of dilatation alone, the paroxysms of palpitation and dyspnœa in particular being more violent, more obstinate, and more easily excited.

b. Diseases of certain valves impress well-defined peculiarities on the pulse. This subject has been very imperfectly understood, and it is therefore necessary to warn the student against the erroneous statements which he will find in various authors. The original genius of Corvisart attempted to connect peculiarities of the pulse with diseases of the heart; but, destitute of the light of auscultation, he signally failed in the particular applications. The illustrious father of auscultation, seeing the failure of Corvisart, seems to have abandoned the attempt. He devotes nearly a whole chapter to proving that "the exploration of the pulse is far from being able to give an idea of the *general* circulation, and cannot make known the manner in which it is carried on even in the heart" (vol. ii. p. 473—9). He seldom, therefore, mentions the pulse, except as a quotation from Corvisart and others, to display its fallaciousness. M. M. Bertin and Bouillaud believed that, in contraction of the valvular orifices, the pulse was valueless as a sign, quoting Corvisart to display how totally he was deceived in it, and contending that the auscultatory signs of Laennec were the only indications of valvular disease worthy of confidence (Traité, p. 225). Dr. Elliotson, in 1830, depreciates the pulse and upholds auscultation, like the three preceding authors (Lumleyan Lectures, p. 17 and 27). M. Bouillaud, in his treatise in 1835, (ii. p. 217,) *makes a single description of pulse answer for the whole of valvular diseases!* He says, closely following Corvisart, "The pulse, irregular, unequal, and intermittent, contrasts by its smallness, its *minuteness* (exiguité), with the energy, violence, and extent of the beats of the heart: notwithstanding its smallness, it is *hard* and vibrating when the contraction is attended with great hypertrophy of the left ventricle." Now, it is obvious that a single variety of pulse cannot answer for every variety of valvular disease; besides, *hardness* and *extreme minuteness* are absolutely incompatible qualities!

The mistakes of the whole of these authors have originated in their unacquaintance with particular valvular diagnosis, whence they mistook the pulse of one valve for that of another.

I have been endeavouring, since the year 1823, to supply the deficiencies to which I allude; but it is not until lately that the subject has admitted of being brought to a satisfactory conclusion, as discoveries on the pulse could only be consecutive to a succession of other discoveries, which, up to the present time, have gradually been giving additional precision and certainty to the diagnosis of cardiac diseases. During the last four and a half years, I have made written notes of the pulse in 10,000 cases. The limits of this work do not permit me to give more than the general results of these and my previous researches.

The pulse in disease of the mitral valve.—When the mitral valve is contracted, and also when it admits of free regurgitation, the pulse is, in various degrees, small, weak, irregular, intermittent, and unequal. When either the contraction or the regurgitation is great, the whole of these characters are invariably present, as in the cases of Dolan, Dennis, Anderson, Sharpe. But when the degree of either is slight, (when, for instance, the circumference of the orifice is not diminished more than an inch, or when the aperture for regurgitation is not larger than a goose-quill,) the effect on the pulse may only be a slight degree of weakness and intermittence, increasing when the circulation is hurried.

The explanation of the pulses in question I conceive to be as follows. In the case of contraction of the mitral orifice, the left ventricle, not being freely supplied with blood, is not stimulated to contract at the natural intervals, with suitable energy, and in equal degrees. In the case of regurgitation, the ventricle, having lost the resistance of the mitral valve, expends the force of its contraction in the retrograde, as well as in the forward direction, and also expels into the aorta a diminished quantity of blood; whence the pulse is proportionably feeble and small: further, as the regurgitation disturbs the regularity of the supply to the ventricle, more or less of intermittence, irregularity, and inequality are sooner or later the result.

It may here be well to explain, that intermittence is the least degree of derangement of the heart's action, as its rhythm is not subverted, there being only the occasional omission of a beat, the next beat recurring at the regular interval. Irregularity is an ulterior degree of derangement; for, here, the rhythm *is* subverted, the beats recurring at irregular intervals. Inequality almost always accompanies irregularity, some beats, both of the pulse and heart, being stronger than others; and I have frequently noticed a stronger beat to be followed by one, two, or even three weaker ventricular contractions, audible by the stethoscope, but scarcely, and sometimes not at all, sensible in the pulse.* When one or two beats are regularly and permanently imperceptible in the pulse, such cases constitute the bulk of those in which the pulse is described by non-auscultators as being singularly slow,—for instance, 30 or 20 per minute. In a few rare cases, however, it is really slow. I have lately seen three instances in which it was as low as 28, without any intermediate ventricular contractions. In one, there was no disease of the heart, and the patient completely recovered, hypercatharsis after fever having been the cause. I have repeatedly seen the pulse at 40, from mere depression of the nervous system.

Certain other affections, besides disease of the mitral valve, may render the pulse small, weak, intermittent, irregular, and unequal. These exceptions and their diagnosis must, therefore, be briefly noticed. 1. Softening of the heart, as already shown, (p. 339) may occasion all the above characters of the pulse in the highest degree: it may be known by the absence of valvular murmurs. When softening coexists with mitral disease, the two will, of course, co-operate in producing the pulse in question. 2. The same pulse may be produced by pericarditis with copious effusion compressing the heart, by endocarditis causing polypi in the cavities (Bouillaud), and by polypus in any other disease of the heart. These diseases may be severally known by their own characteristic symptoms, and by the sudden supervention of the state of pulse. 3. Dyspepsia, nervousness, biliousness, and gout may respectively occasion several, or all of the above qualities of the pulse: they may be known by the attacks of irregularity being only occasional and temporary, and by the absence of valvular murmurs.*

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* M Bouillaud is mistaken in supposing that he was the first to notice this species of irregularity. It was described in the first edition of this work, p. 332. He has ascribed the weaker sounds and impulses to wrong causes, as already shown (p. 64 and 272, note).

The pulse in contraction of the aortic valves.—Contraction of the aortic valves must be very great to render the pulse small, weak, intermittent, and irregular. I have never seen it possess these characters in any marked degree, unless the valves were either soldered together by cartilaginous degeneration, (case of Hedgley,) or more or less fixed by ossification in the closed position, so that the aperture was only a limited chink. An induration of the size of an ordinary pea has little effect on the fulness, firmness, and regularity of the pulse, (cases of Porter and May,) and slighter degrees of contraction appear to have no effect on it whatever. I have proved this by cases published in the Med. Gaz. Sept. 1829, and could corroborate it by a great number more. It is obvious, indeed, that as the *supply* of blood to the left ventricle is regular, its action must partake of that regularity, and that, when the contraction of the aortic valves is not so great as to prevent the ventricle from emptying itself, the pulse will remain full and firm.

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* Upwards of a year previous to my discovery of mitral regurgitation and its pulse, in June 1825, I had noticed the pulse of mitral contraction, and given an account of it, recorded in the Essays of the Roy. Med. Soc. of Edin. for 1824. Mr. Hodgson, I have since found, had previously observed that, in mitral disease, there was often a "double pulsation of the heart," one of which pulsations he incorrectly ascribed to the contraction of the auricles. Since 1824, the pulse of mitral contraction has been noticed by Mr. Adams (Dub. Hosp. Rep. iv. p. 420); Dr. Elliotson, quoting Adams; and Dr. Hodgkin (Med. Gaz. vol. iii. p. 448), who was a fellow-student of mine in the Edin. Infirmary and Royal Med. Soc., where he possibly imbibed the idea.

It was respecting the pulse of aortic contraction that Corvisart made his principal mistake; and, as it has been copied by almost every subsequent writer, it requires a moment's consideration. "The pulse," says Corvisart, "may retain a certain degree of hardness and tension, but *never much of fulness or regularity.* This *invariable and permanent irregularity* will always be sufficient to furnish a precise diagnosis of contraction of the aortic orifice." Louis follows Corvisart (on Pericarditis, p. 12). Bouillaud falls into the same error in the only instance in which he connects the pulse with the particular valve diseased. "The pulse," says he, "is in general more irregular, small, unequal and intermittent in simple contraction of the aortic orifice, than of the mitral (Traité, 1835, ii. p. 221). Now, except in the very few cases of extreme aortic contraction, this is nothing more than the pulse of mitral contraction or regurgitation; for, by abstracting all the cases of these latter affections, (which neither Corvisart, Louis, nor Bouillaud were competent to do, because they were strangers to particular valvular diagnosis,) I have ascertained in the most positive manner that the characters of *smallness,* and *invariable, permanent irregularity,* are totally foreign to the pulse of aortic contraction, when not extreme. If it be urged that the above authors, though strangers to particular valvular diagnosis, could ascertain by autopsy which was the valve diseased, in correspondence with the pulse in question, a negative rejoinder may be given; because, before the regurgitations were discovered, various lesions of the auricular valves occasioning them, (e.g. mere shortening of the chordæ, inflammatory adhesion of the posterior fold of the valve, atrophy of the valve, &c.) were totally and habitually overlooked.

The pulse in regurgitation through the aortic valves.—Under this head must be included regurgitation out of the aorta into the right ventricle (Mitchell), or into the pulmonary artery (Evans). Aortic regurgitation produces a pre-eminently *jerking* pulse, a high degree of the pulse of unfilled arteries, as seen in anæmia from any cause. The diastole or beat of the artery is short and quick, as if the blood were smartly jerked or shot under the finger, the vessel during the intervals feeling unusually empty. This is the most remarkable, appreciable, and constant pulse produced by disease of the heart. In the immense majority of cases, the practitioner may conjecture the disease by this sign alone. It differs from the jerking pulse of anæmia, in being more marked, and in not necessarily being frequent, as the anæmic pulse is, when its jerk is distinct. It may be absent, or scarcely appreciable, if the regurgitation be very slight; and it may be neutralized by free mitral regurgitation (Payne) or great contraction, in consequence of the enfeebling effects of these lesions on the pulse.[*]

Valvular diseases of the right side of the heart produce little effect on the pulse; 1. because there is not a direct connexion between that side and the arterial system; and, 2. because the action of the organ is less under the influence of the right ventricle than of the left, in consequence of the superior muscular strength of the latter. Fortunately, as valvular diseases on the right side are so rare, we stand less in need of the evidence afforded by the pulse. In reference to the left side, that evidence is of great value; for it not only substantiates the physical signs, but sometimes indicates the degree of a valvular disease, while the physical signs merely announce the fact. Thus, a decidedly jerking pulse denotes a free aortic regurgitation, and a decidedly weak and irregular pulse bespeaks great mitral contraction or free regurgitation. Nor is this knowledge a mere diagnostic refinement. It is of practical value; since a latitude may be permitted to the patient in slight degrees of the disease, which would be totally inadmissible in the more advanced.

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[*] I described this pulse, (which had not previously been noticed by any writer on diseases of the heart,) in several parts of the first edition of the present work in 1831; especially at p. 434; but, having up to that time noticed it solely in cases of aortic regurgitation combined with inflammation of the heart or adhesion of the pericarrium, I was in doubt as to its cause, and ascribed it more to the latter affections than to the regurgitation, propounding, however, the question, in reference to the case of Copes, written in 1829, whether it was not due to the regurgitation. This question I soon after resolved in the affirmative by discovering the pulse in question in cases of the regurgitation alone. Dr. Corrigan, who wrote in 1832 or 1833 on permanent patency of the aortic valves as a supposed new disease, has so completely overlooked this pulse as even to state the reverse: "it rises without any jerk under the finger" (Dublin Jour. vol. x. p. 186). M. Donné subsequently wrote a thesis on aortic regurgitation, which I have not been able to procure; but as M. Bouillaud, who quotes him, does not anywhere allude to the jerking pulse, I presume that it was overlooked by M. Donné also.

c. Pain in the region of the heart is another symptom that affords presumptions of disease of the valves. It is true that palpitation or engorgement of the heart may occasion pain, though there be no disease of the valves: I have frequently met with it from these causes in hypertrophy and dilatation. It is likewise true that palpitation may occasion pain, though there be no disease of the heart whatever; I have found it in a large proportion of hysterical, anæmic females and nervous males. But it is when the valves, the coronary arteries, or the commencement of the aorta, are indurated and inelastic, that pain occurs most frequently and with the greatest severity. Sometimes it is little more than an indescribable sense of obstruction or oppression in the præcordial region; but, in other cases, it is an intense lancinating or tearing pain, felt across the præcordia or scrobiculus cordis, (where it might be mistaken for inflammation of the stomach,) and occasionally extending, with a sense of numbness, down the inside of the left arm to the elbow, and sometimes to the fingers. Pain of this description has acquired the name of *angina pectoris.* (See Angina Pectoris.)

I believe this pain to be, in general, occasioned by the inelasticity of the ossified or otherwise indurated parts, which will not stretch equally with the other portions of the heart, when the organ is labouring under palpitation or engorgement. When inflammation of the interior of the heart exists, it also may occasion pain; but those authors have unquestionably been wrong who have considered inflammation to be the *sole* cause of pain, and have therefore assumed this symptom as proof of the inflammatory nature of disease of the valves. The truth is, that the pain of acute endocarditis is neither of common occurrence, nor considerable in degree.

Progress, terminations and prognosis.—The exact time and manner of the fatal termination in valvular disease, as in every other organic affection of the heart, is very uncertain. Sometimes the patient is reduced gradually to an extreme degree of emaciation and debility, and dissolution is duly announced by the usual premonitory symptoms. Sometimes he expires suddenly, after any exertion or emotion, though the malady have made comparatively little inroad on the constitution. In this case the event must be attributed to the obstruction having attained, by the progress of the disease, such a point that the heart, when hurried beyond a certain degree, can no longer maintain the circulation against it. Not unfrequently, pressure on the brain, whether from venous congestion, or its consequence serous effusion, is the immediate cause of death, and in this case coma usually supervenes gradually, in the course of from three to four days or a week previous to the fatal event (Dolan). It may, however, occur abruptly. In one case, under my care, of serous effusion, the patient suddenly uttered a shriek and fell at once into perfect coma; and I have seen many cases of sudden apoplexy, both congestive and hæmorrhagic.

Hence, the prognosis must always be general as to time, and, if the case be considerably advanced, it must be guarded with a clause, that the patient is liable to die suddenly and unexpectedly. This catastrophe, however, is much more rare since the improved diagnosis of the diseases of the heart has made it possible to enjoin suitable precautions.

Physical Signs. — Before the discovery of auscultation, it was extremely difficult, and in many cases utterly impossible, to detect disease of the valves. Corvisart had the merit of discovering, as its signs, certain states of the pulse, and a "peculiar vibration difficult to describe, sensible to the hand applied to the præcordial region:"—in other words the cat's *purring tremour* (*frémissement cataire*) of M. Laennec. But, as these signs may occur under other circumstances, they do not denote disease of the valves in particular, and are totally insufficient to indicate which is the valve affected. The accession of auscultation to the other means of diagnosis, has rendered it possible to distinguish valvular disease, both in general and in particular, with almost complete certainty: a certainty, it may be remarked, much greater than was supposed by the illustrious author of auscultation himself; for he did not give their full value to preternatural murmurs as signs of disease of the valves, in consequence of supposing that similar murmurs were produced by spasmodic contraction of the muscular fibre of the heart and even of the arteries, and in consequence of being unacquainted with the whole class of murmurs from regurgitation subsequently discovered by the writer. Thus he says, "The anomalies in the sounds of the heart and arteries of which I am going to speak (viz. *bruit* *de soufflet* and *frémissement cataire*) are the more remarkable, because, of all those which auscultation has revealed, they alone are not connected with any organic lesion in which we can find their cause:" and again, "It appears to me that the positive and negative facts which I have just advanced, all tend to prove that *bruit de soufflet is the product of a simple spasm, and does not suppose any organic lesion in the heart and arteries.*" I have shown, at p. 95 et seq., that it is not spasm of any kind, but a modified movement of the blood, attended with increased friction and vibration, which is, in all circumstances, the cause of inorganic murmurs and tremors, whether in the heart, the arteries, or the veins, and whether ordinary, continuous, humming, or whistling. Independent of these, there are no murmurs and tremours which may not be distinctly traced to organic causes. Laennec laboured under another disadvantage: he attributed the *second* sound of the heart to the auricular contraction; whereas, it is demonstrated by the experiments of the writer that the auricles yield no sound, (p. 24,) and that the second sound is referable to the closure of the semilunar valves (p. 50): also, that the *first* sound, instead of being wholly muscular, as he imagined, is partly muscular, but principally valvular (p. 50). These errors necessarily perplexed him in referring murmurs to their true source.

Having, in the section on *murmurs from valvular disease*, (p. 81 et seq.,) fully considered the nature, causes and mechanism of the *bellows*, *filing*, *sawing*, *rasping*, and *musical* or *whistling* murmurs; having pointed out the situations where they are to be explored, (p. 90,) and having, in the section on *purring tremour or thrill*, (p. 124,) presented a synoptical sketch of this associated phenomenon; I now proceed to show in what manner they constitute signs of disease of each of the valves in particular. It may be premised that, as the sounds of one side of the heart are audible on the other, the sound of the healthy side will partake somewhat of the murmur of the diseased side; yet not so as to create a fallacy, if due attention be paid to the diagnostic rules which will now be offered.

*Signs of Disease of the Aortic Valves.**—One of the mur-

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* The young student is strongly recommended to copy the diagrams Fig. 4, and carry them about with him in his pocket, till he is thoroughly master of this subject.

murs above alluded to is heard during the ventricular contraction, (i. e. with the first sound,) on the sternum, opposite to the lower margin of the third rib, and thence for about two inches or more upwards, along the course of the ascending aorta towards the right; and it is louder in these situations than below the level of the valves. Its pitch or key is usually that of a whispered *r*, from being superficial, and it accordingly conveys the idea of being pretty near to the ear. When a murmur of this kind is considerably louder along the tract of the ascending aorta than opposite to its valves, and is, at the same time, unusually near-sounding and superficial—in other words, on a higher key than a whispered *r*, it proceeds from disease of the ascending aorta itself. As the murmur from this cause is audible in the situation of the valves, it might lead to the supposition that they also were diseased, and it is sometimes very difficult to ascertain positively that they are not. That a murmur is seated in the aorta, and not in the pulmonary artery, may be known by its being inaudible or very indistinct high up the course of the pulmonary artery, while it is distinct high up that of the aorta. That a murmur is seated in the aorta or its valves, and not in the auricular valves, may be known by its sounding loud and *near* above the aortic valves, where an auricular murmur, if audible at all, sounds feeble, *remote*, and on a low key, like a whispered *who.*

When there is regurgitation through the permanently open aortic valves, a murmur accompanies the second sound, and its source may be known by the following circumstances:—1. It is louder and more superficial opposite to and above the aortic valves than about the apex of the heart, by which it is distinguished from a murmur in the auricular valves with the second sound. It is louder along the course of the ascending aorta than along that of the pulmonary artery, and down the tract of the left ventricle than down that of the right; by which circumstances its seat is known to be in the aortic, and not in the pulmonic valves. This inference is strongly corroborated by the state of the pulse, which, when the aortic regurgitation is at all considerable, is singularly and pre-eminently jerking—the pulse of unfilled arteries. 3. It is distinguished from a systolic murmur in the aortic orifice by its accompanying the second sound; by its being more audible, (though with a gradual diminution,) down the course of the ventricle, than a systolic murmur; by its being prolonged through the whole interval of repose, and even through accidental intermissions of the ventricular contraction (case of W. Esq.); and by the weakness of the refluent current always imparting to it the softness of the bellows-murmur, an inferior degree of loudness, and a lower key, like whispering the word *awe* during inspiration. It often becomes musical.

Purring tremor, though necessarily produced by any considerable, salient, or rugged contraction of the aortic valves, can rarely be felt, because the sternum is interposed; but when the heart is displaced from beneath the sternum, as by hydrothorax, empyæma, emphysema, tumours, consolidation and contraction of one lung and hypertrophy of the other (case of James), &c. the tremor may then become perceptible (case of Mitchell). I have never known it accompany aortic regurgitation.* Probably the refluent current is too feeble to render it perceptible through the walls of the chest. Aortic regurgitation, however, by unfilling the arteries, eminently favours the production of tremor from contraction of the aortic valves, during the ventricular systole (see p. 126).

Irregularity of the pulse is not necessarily or usually produced by contraction of the aortic valves, unless extreme (e. g. case of Hedgley); nor are the size and strength of the pulse materially diminished by moderate contraction. Aortic regurgitation produces the eminently jerking pulse; and this it does whether the regurgitation be into the left ventricle, or through a false opening into the pulmonary artery or mouth of the right ventricle (Mitchell and Evans).†

Signs of Disease of the Pulmonic Valves. — The signs of contraction of the pulmonic valves are the same as those of the aortic, (p. 383,) with this difference; that, from the vessel being nearer the surface, the murmur with the first sound seems *closer* to the ear, and is on a higher key, ranging from the sound of a whispered *r* towards that of *s*. I have, however, known it fall below *r* when the circulation was feeble and slow, and the obstruction slight. It may be known that the murmur is not seated in the aorta, by its being inaudible, or comparatively feeble, two inches up that vessel; whereas, at a corresponding height up the pulmonary artery, it is distinct: also, by its being louder down the tract of the right ventricle than down that of the left (Bowden). It may be known that the murmur does not proceed from regurgitation through the auricular valves, by its being distinct along the course of the pulmonary artery, where auricular murmurs are either wholly inaudible, or very feeble and remote.

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* It did not exist even in a case of displacement which I lately examined, where the aorta beat between the second and third ribs, an inch to the right of the sternum.

† While I was writing the above, a physician brought to me his son, affected with aortic regurgitation, who, he said, had what a friend called a "*quick, slow pulse.*" The epithet is quaintly expressive. It is rendered in Latin by *celer et infrequens*; whereas the jerking pulse of anæmia is *celer et frequens.*

When a murmur in the pulmonary artery is considerably louder between the second and third left ribs, close to the sternum, than opposite to the valves, and is there attended with impulse and purring tremor, dilatation of the pulmonary artery may be suspected (see *Dilatation of Pulmonary Artery*). In one instance I have known a murmur to be produced by complete ossification of the pulmonary artery penetrating deeply into the lungs (case of Lady R.).

When there is regurgitation through the pulmonic valves, a murmur accompanies the second sound. Its nature and diagnosis are the same, (the necessary inversions being made,) as in the case of aortic regurgitation, (p. 384,) except that the pulse is not jerking (case of Rogers. A tremor attended).

I presume that purring tremor with the first sound may be occasioned by contraction of the pulmonic orifice, though I have not met with an instance verified after death: but I have met with three in which the tremor attended dilatation of the pulmonary artery (Weatherly, Bowden, and Miss L. P——r). A purring tremor occasioned by the pulmonic valves would be more readily felt than one occasioned by the aortic valves, because it would probably be transmitted as far as the space between the second and third ribs, (where it is out of the cover of the sternum,) provided the patient lay in the horizontal position, and inclined to the left side.

Disease of the pulmonic valves is so rare, that it ought never to be suspected unless the signs described are perfectly well marked, or unless there be patescence of the foramen ovale, or some other communication between the two sides of the heart,—states which experience has proved to be generally accompanied with contraction of the orifice in question.

Signs of Disease of the Mitral Valve.—When the valve is permanently open, admitting of regurgitation, the first sound is attended with a murmur. It may be rough, (rasping,) or smooth, (bellows-murmur,) according to the nature of the contraction, &c. (p. 83). Its key is low,—more or less like whispering *who* (p. 86); yet it sounds loud and *near* if explored about the apex of the heart, and a little to the sternal side of the nipple.* It may thus be easily distinguished from a direct semilunar murmur, which, in this low situation, always sounds feeble and *distant.* The murmur in some cases completely drowns the natural first sound on the left side: in others, the sound can be distinguished at the commencement of the murmur.†

I have found perceptible purring tremor to be produced more frequently by regurgitation through the mitral valve than by any other valvular lesion—especially when the ventricle was hypertrophous and dilated, by which the refluent current was rendered stronger.

If the regurgitation be considerable, but not otherwise, the pulse is more or less small, weak, intermittent, irregular and unequal (p. 376); and this, even though the impulse of the heart be violent.

When the mitral valve is considerably contracted, a murmur (best heard in the same situation as the murmur from regurgitation, and distinguishable in the same way from semilunar murmurs,) attends the ventricular diastole and second sound. From the weakness, however, of the diastolic current out of the auricle, the murmur is always very feeble, soft like the bellows-sound, and usually on a rather lower key than a whispered *who* (p. 86). I have found this murmur absent unless the contraction of the valve was considerable; for the blood had still sufficient room to pass with tranquillity: and I have also found it absent when the contraction was *great*—when, for instance, the aperture admitted one finger only, or merely a quill, provided the current was preternaturally weakened by softening, by extreme dilatation of the heart, or by both (cases of Anderson and Mrs. ——l—n). In such cases, however, the mitral disease would not be overlooked, as there is almost invariably a murmur from regurgitation. On the whole, this murmur is exceedingly rare, though Laennec and authors in general have supposed quite the contrary, from mistaking for it the murmur of aortic regurgitation (see p. 78).

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* The reader is particularly requested to refer to Figs. 2 and 3, where he will distinctly see the principle on which the sounds, both natural and morbid, of the auricular valves are transmitted to the apex of the heart, rather than to the anterior walls, opposite to the auricular orifices.

† The natural sound must not be confounded with the accidental sound of costal percussion, and *metallic tinnitus*, produced, as I have shown, (p. 41,) by the apex of the heart impinging against the lower edge of the fifth rib, as it glides up during the systole; and occurring in emaciated, anæmic subjects during palpitation. Thus, in the case of Jones, a murmur superseded the natural first sound; but, during palpitation, the sound of costal percussion became loudly perceptible.

I have never known purring tremor accompany a diastolic mitral murmur, the current being too feeble to produce it.

When the contraction of the mitral valve is great, the pulse (whether there be regurgitation or not) is more or less small, weak, intermittent, irregular and unequal, in consequence of the supply of blood to the left ventricle being insufficient and irregular (p. 376). I have known the same to be occasioned by a polypus choking up the left auricle.

Signs of Disease of the Tricuspid Valve. — They are the same as those of the mitral, except that the murmurs are loudest on or near the sternum, at the same level as in the case of the mitral—namely, about or a little above where the apex beats; and except, also, that the pulse is little affected with irregularity. I have never known purring tremor produced by this valve.

As the tricuspid valve is very rarely so much diseased as to yield a murmur, its lesions being exceedingly unfrequent and almost always in a slight degree, the practitioner must be very cautious in pronouncing it diseased, especially as the pulse does not afford the same evidence as in contraction of the mitral orifice.

Signs of Disease of the Arterial and Auricular Valves conjointly.—The murmurs above described as characteristic of each, exist simultaneously in both. The auscultator has merely to take especial care that he explores the arterial murmurs as high up the vessels, and the auricular murmurs as low down the heart, as possible. He will thus readily satisfy himself that there are two distinct sources of murmur. It is still easier to determine this, if the murmur attending either sound be of a different *species* in the two situations—if, for instance, the murmur of the aortic or pulmonic valves be of the *soft* bellows-kind, while that of the auricular valve is of the *rough*, grating or rasping kind, or *vice versâ*.

Diagnosis of Valvular from Inorganic Murmurs.—To make the above signs completely available, it is necessary to attend to several circumstances which might lead to deception. Bellows-murmur, as already fully explained, (p. 98,) sometimes exists in the heart, though there be no disease of the valves: namely, in anæmic persons, who, at the same time, are generally nervous and excitable (p. 102); in excessive hæmorrhage and the reaction following it, where anæmia is still the essential cause (p. 100); and in a very few cases of hypertrophy with dilatation, where again the phenomenon is principally anæmic (p. 94). Murmur from these causes may easily be distinguished from that of valvular disease by the following criteria.

1. It is confined to the aortic orifice, (so far as I have yet discovered,) and to the first sound. Here is one of the great advantages of *particular* valvular diagnosis, as the auscultator can at once exclude the other seven murmurs to which the heart is liable from organic causes only.

2. *It is always weak, and of the soft or bellows kind.*

3. In the anæmic, it is almost invariably attended with a continuous venous murmur in the jugulars, and mostly with a short bellows-whiff, in the carotids, subclavians, and other principal arteries, synchronous with the first sound of the heart.

4. It exists in the anæmic during temporary excitement of the circulation only, subsiding when palpitation ceases and the pulse falls to its natural standard; but as the pulse is permanently quick in considerable anæmia affecting irritable, nervous subjects, especially females; also during the period of reaction after excessive loss of blood, the murmur will persist until the pulse falls by the subsidence of the states in question.

5. The murmur, both of the heart, arteries, and veins, wholly

ceases when the anæmia is cured by iron and animal food, the venous murmur being the last that becomes extinct.

When a murmur proceeds from hypertrophy with dilatation, it may be known by its diminishing or ceasing when the action of the heart is calmed, as by repose, venesection, digitalis, &c. In most, if not all cases, this murmur is dependent merely on anæmia, which is very apt to supervene in the advanced stages of hypertrophy with dilatation.

Contrasted with the above, the distinctive characters of valvular murmurs are, 1. That they are not, like inorganic murmurs, restricted to the aortic orifice and first sound, but may be connected with any of the four orifices and with either sound in each: 2. That they persist without intermission for an indefinite length of time, even though the heart be kept in a state of perfect calm: 3. That they are often of a *rough* character, that is, filing or rasping; whereas, inorganic murmurs have always the softness of the bellows-sound.

Such are the signs which, together with the general symptoms, are, according to my experience, the best for the detection of the diseases of the valves. In the first edition of this work, where the signs were less fully developed, I was enabled to say that "for several years they had never deceived me as to the general fact whether there was or was not valvular obstruction, and that they had seldom failed to indicate, with perhaps more than necessary precision, the situation of the affection." I may now venture to add, that, with the improvements introduced in the present edition, the particular diagnosis is even more easy and certain than the general; because a practitioner competent to make the latter only, is more liable to be deceived by inorganic murmurs. I have no doubt that, ere long, the physical signs in particular will be universally admitted to be as simple and easy as I represent them to be, since I have found by trials that intelligent students are competent to make particular diagnosis after a verbal explanation not exceeding a quarter of an hour's duration. I feel assured also that valvular diagnosis will shortly be acknowledged to be more certain than that of the muscular diseases of the heart, because the physical signs of valvular disease are more fixed.

If it be said that particular valvular diagnosis is a useless refinement, it may be replied that non-auscultators used to say the same of auscultation in general. The truth is, that every improvement in diagnosis is an advantage to the practice of medicine. No one, for instance, will deny the importance of distinguishing inorganic from organic murmurs, as the treatment for the two is diametrically opposite; and this distinction, it has been shown, is remarkably facilitated by particular valvular diagnosis. Again, the pulse, without particular diagnosis, is unintelligible even to the most learned, as Corvisart, Laennec, &c., and has betrayed them into grievous practical errors. Further, disease of certain valves is more injurious and dangerous than that of others. Unless, therefore, the practitioner is able to specify the valve diseased, he cannot nicely adapt his treatment to the exigencies of the case, but must in some instances be uselessly rigid, and in others dangerously lax.*

As an appendix to the present subject, I may advert to a few unusual and curious sources of murmur independent of valvular disease, which constitute the only remaining causes of fallacy with which I am acquainted.

1. I had a patient in the St. Mary-le-bone Infirmary, in whom I, as well as the apothecary Mr. Hutchinson, noticed a distinct murmur along the ascending aorta on some occasions, and not the slightest on others. I was much perplexed, and could not make up my mind as to the existence of valvular or aortic disease. The patient died of phthisis, and on post-mortem examination, it was found that the anterior edge of the left lung, com-

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* It is astonishing that a writer who has had so much experience as M. Bouillaud, does not even pretend to particular valvular diagnosis so late as the year 1835. The following is his summary of signs: "To sum up, when we hear in a patient a permanent bellows, rasping, or sawing murmur in the præcordial region, when there is at the same time a vibratory tremor and palpitations, or tumultuous, irregular, intermittent beats of the heart, it is almost certain, if the disease is already of several months or years standing, that there is an induration of the valves with contraction of one or several orifices of the heart. Nothing is wanting to the certainty of the diagnosis when, to these local signs accede the signs called general, and which are the result of the influence exercised on the functions of the other organs by the obstacle to the passage of the blood through the heart" (Traité, ii. p. 216). Most assuredly, no one could mistake a valvular disease which presented all these signs; but the great majority do not present half of them! What is to be done then? It is no wonder that valvular diagnosis was difficult while the signs were so complex, vague, and general, as M. Bouillaud makes them.

pletely indurated by tubercular deposition, pressed so exactly on the ascending aorta as actually to have taken its mould, though without adhering. It was now recollected that the murmur had always been heard when she lay on her back or inclined to the right side, but not when inclined to the left: hence we ascribed it to pressure of the lung on the aorta when the position of the body caused it to gravitate towards the right side.

2. Two students of University College called on me, one with slight hypertrophy with dilatation, and violent palpitation from great nervous excitability,—the pulse, for instance, being 120; the other was exempt from organic disease, but affected with violent nervous palpitation, the pulse here also being 120. Both wore very tight waistcoats, preventing the expansion of the lower ribs. During this state of breathing, with the lungs insufficiently inflated, a slight bellows-murmur with the first sound over the semilunar valves existed in both. It was not, however, *exactly* synchronous with that sound, but began an instant later, as if from a separate cause. In both, the murmur ceased *entirely* when, unbuttoning their waistcoats and waistbands of their trowsers, they breathed with the lungs naturally inflated. By alternating the circumstances, the murmur could be created or removed at pleasure. I presume, therefore, that it proceeded from a cause exterior to the heart; and, as the murmur was an instant later than the first sound, the most probable appears to be, that, in the contracted state of the chest, the violent beats of the heart compressed the lung, and, by suddenly expelling its air, created a murmur.

3. Dr. Elliotson mentions two or three cases somewhat analogous. In one—a case of ascites—a bellows-murmur with the first sound, in the region of the left ventricle, instantly ceased on the removal of the fluid from the abdomen; but when it re-accumulated, the sound again became audible. In another case—a young woman with chronic bronchitis, dyspnœa, livid lips and œdematous legs—no murmur existed while she was erect, but it became audible the moment she lay down (Lum. Lecs. p. 18). Dr. Elliotson conjectures that, in the first case, the elevation of the heart by the abdominal fluid might have tilted the organ to

an angle with the commencement of the aorta: and, in the second case, he thinks that the cessation of the murmur when the patient was erect, depended on the ventricle being, by gravitation, drawn down more into a straight line with the aorta, when an easier exit was given to the blood. Another conjecture in which he indulges, but to which I cannot assent, is, that dilatation of the right auricle, by pressing against the aorta, might have occasioned the murmur in both.

I have not data by which to decide these points, but the practical inference is, that, in cases of slight bellows-murmur with the first sound, and connected with the arterial orifices, (for the fallacy cannot apply under any other circumstances,) we should not decide till we have ascertained that the murmur continues in the erect as well as the recumbent position, and also while the chest is totally unrestrained by ligatures. I have at present a case of an exceedingly anæmic girl, æt. 17, in whom a venous murmur in the vena innominata was propagated down the great vessels, especially the pulmonary artery, and led a young auscultator into the error of supposing that there was disease of the pulmonic valves.

In conclusion, these anomalous cases are very rare; and they will create little difficulty, if due attention be paid to the rules laid down respecting the best situations in which to explore the murmurs of the several valves.

SECTION V.

CARDIAC ASTHMA.

Amongst the diseases of the heart may be justly reckoned one of the forms of the malady termed in common language *asthma*. This has been too much regarded as independent of disease of the heart. Long treatises have even been written upon it without ever mentioning disease of this organ as one of its causes.

It is, therefore, necessary to notice the subject formally in this place, not only for the purpose of showing the magnitude of the error, but of making the reader acquainted with all the habitudes and aspects of a complaint, which is perhaps the most distressing in the whole catalogue of human maladies.

It is established by the concurrent testimony of all moderns conversant with diseases of the heart, that these diseases, no less than those of the lungs, may constitute the organic causes of asthma.

A theoretical consideration of the subject leads, in my opinion, to the same conclusion; for, on tracing asthma back to its source, we shall find that, whatever be its proximate cause in different cases, it is connected, in all, with the same ultimate circumstance; namely, inadequate oxygenization of the blood, and the resulting want of breath, which, through the "incident excito-motory" branches of the pneumogastric, excites the "reflex" action of the "true spinal" nerves on the muscles of respiration. For instance, inadequate oxygenization of the blood results in all ordinary cases from one or more of three proximate causes: viz.

A. *Insufficient admission of air into the bronchial tubes and air-vesicles.*

B. *Insufficient exposure of the blood to the air admitted, in consequence of a less pervious state of the mucous membrane than natural.*

C. *Insufficient admission of blood into the lungs.*

It will be found that, to one or more of these causes, all the varieties of dyspnœa and asthma are referable.

All the varieties of asthma—to give an approximative statement probably very near the truth—are comprised under the following heads:—

1. From *chronic dry catarrh*, and the emphysema resulting from it.

2. From *pituitary catarrh* (humoral asthma) whether acute or chronic, but more especially the latter, and the pulmonary œdema resulting from it.

3. From *mucous catarrh*, especially chronic.

4. From *organic disease of the heart.*

5. From purely *spasmodic constriction* of the bronchial tubes.

I do not include amongst the varieties, one from the compression of the lungs by hydrothorax, by tumours, by imperfect descent of the diaphragm, &c., because these rarely occasion what can strictly be called asthma. Before examining the above varieties it may be premised that, whatever be the organic cause, all suppose the superaddition of bronchial spasm, as will be explained under the fifth variety.

1. *Chronic dry catarrh* is attended with intumescence of the internal membrane of the bronchial tubes. The intumescence exists principally in the smaller tubes, which are sometimes completely obstructed by it; but it is also found in the larger. Andral has seen the bronchial trunk of a lung so contracted by this intumescence, that the air could scarcely enter; and in another case, the third and fourth bronchial divisions were contracted by the same cause (Clinique Med. seconde partie, obs. ii. et iii.). Further, the tubes are more or less obstructed by an exceedingly viscous mucus, often as dense as the vitreous humour of the eye; and when the dry catarrh is universal or very extensive, it is almost invariably productive of emphysema.

2. *Pituitary catarrh* is attended with moderate intumescence, slight softening, and partial redness of the pulmonary mucous membrane—a state intermediate between sanguineous and serous congestion, but partaking more of the latter. The quantity of phlegm expectorated, always considerable, is sometimes enormous, amounting to from four to six pints of thin glairy fluid in twenty-four hours.

The air-passages being obstructed partly by the intumescence of their mucous membrane and partly by this fluid, it necessarily follows that there is an insufficient admission of air into the lungs.

3. *Mucous catarrh* is accompanied with more or less tumefaction of the bronchial membrane and obstruction of the calibre of the tubes. The expectoration, though less copious, and different in quality from that of pituitary catarrh, is, notwithstanding, frequently abundant, amounting to one or two pints or more in the day. Consequently, there is an insufficient ingress of air into the lungs.

In all the cases now mentioned, the second cause of inadequate oxygenization of the blood is, likewise, for the most part, in operation; viz. the mucous membrane being thickened, it is less pervious to air; and its mucus, the natural function of which is to expedite the combination of oxygen with the blood, probably discharges this function less perfectly, in consequence of an alteration in its chemical qualities.

4. *Disease of the Heart.*—Sometimes, from this cause, blood exists in the lungs in excess; as is the case when the right ventricle is hypertrophous, or the left side of the heart obstructed; or, still more, when these two affections co-exist: also when the circulation is merely accelerated, as by palpitation, running, or by slighter efforts in corpulent persons. Now, under all these circumstances, there is inadequate oxygenization of the blood; or in other words, there is an excess of venous blood in the lungs: first, because the quantity of blood admitted exceeds its due proportion to the air in the organ; secondly, because the engorgement of the mucous membrane on which the blood ramifies, constricts the bronchial passages, and prevents the free ingress of air, as proved by the feebleness of the respiratory murmur. Hence, want of breath is a necessary consequence of an excess of blood in the lungs.

Sometimes blood does not enter the lungs in sufficient quantity, constituting the third cause of inadequate oxygenization; and this may arise from the weakness of the right ventricle, from an obstruction in its mouth, or from increased resistance on the part of the lungs; as, for instance, during sleep, when the respirative function is less active. Hence results the stimulus of want of breath, and dyspnœa. Cases exemplifying this will shortly be adduced: meanwhile it may be illustrated by a simple physiological experiment, viz. by making and sustaining a full *expiration*. This is attended, not only with a deficiency of air, but also with a deficient influx of blood into the lungs, as is proved by the lividity of the face which ensues, by the elevation of the fontanel in infants; by the rise of blood in a tube inserted into the jugular vein; and lastly, by experiment; for I have demonstrated above, (p. 23,) that, on suspending artificial respiration in a rabbit, the heart *instantly* became gorged, of a black colour, and distended to nearly double its natural size—a phenomenon which renders it sufficiently manifest that, when the lungs are exhausted of air, the blood does not freely enter them. Now, the sensation of want of breath experienced on making a full expiration is familiar to every one, and it becomes intolerable if the expiration be long sustained.

5. *Spasmodic constriction of the bronchial tubes* is presumed to exist, first, because, according to the researches of Reisseissen and others, the bronchial tubes are provided with muscular fibres, and all muscles are liable to spasm: secondly, because asthma is occasionally found to occur without any organic cause (so far, at least, as our senses enable us to judge) sufficient to account for it: thirdly, because every form of organic disease above described, both of the lungs and the heart, may exist without causing dyspnœa of such intensity and of such a character as to constitute *asthma* properly so called. Thus, many have intense chronic bronchitis and profuse expectoration without any asthmatic dyspnœa; and I have known a patient with a contraction of the mitral orifice to the size of a small pea, and likewise with dilatation and softening of the heart and profuse expectoration, pass through a period of ten years to her grave without ever experiencing a paroxysm of asthma, though a few steps across the room were sufficient to excite dyspnœa. (Mrs. —l—n.)

Hence, I apprehend that whatever be the organic cause of asthma, it requires for its production the superaddition of a state of the nervous system leading to spasmodic constriction of the bronchial tubes. Why some should exhibit this state and others not, is one of the arcana of the nervous system; but observation has shown that the state is constitutional and often hereditary.

Admitting that the spasmodic constriction of the bronchial tubes does take place, it is obvious that it will more or less close these tubes against the ingress of air; and this closure, again, by preventing the free expansion of the lungs, will impede the influx of blood. Whence there is a double cause for the inadequate oxygenization of the blood, and, consequently, for the production and maintenance of the asthmatic paroxysm.

From all that has been said, we are now led to the resulting inquiry—what is the essential difference between asthma from disease of the heart and that from disease of the lungs. Putting aside that variety of asthma which, as not being attended with any *visible* organic derangement, (though it is, notwithstanding, highly probable that one exists,) may be regarded as mainly, if not wholly spasmodic, there does not appear to be any essential difference between the remaining varieties. Their organic causes are diversified, but they all ultimately produce the same effect, and it is the effect which constitutes the essence of the disease. This effect is inadequate oxygenization of the blood, which causes "excitant" want of breath; and this, when the case is really asthmatic, i. e. more than what may be called mere dyspnœa, occasions spasmodic constriction of the bronchial tubes, and its consequence, the asthmatic paroxysm.

We now proceed to the more particular consideration of asthma from disease of the heart.

This variety comprises, according to my observation, by far the greater proportion of the most severe and fatal cases of the disease. Some are of opinion that in other varieties the patient experiences an equal degree of suffering during the continuance of the paroxysm. I cannot say that this is consistent with my own observation. Though the same words may suit for the delineation of an attack of each variety, my feeling and conviction is, that I have never seen the patient suffer such intense and suffocative agony as in the variety from organic disease of the heart.

Until the discovery of auscultation had in some degree dissipated the deep obscurity of the affections of this organ, the fact that they were a cause of asthma was scarcely known; and, even at the present day, there are few errors more common than that of attributing asthma to other causes, when it originates solely in the heart. For instance, a theory of this description which has for the last half century been more widely disseminated than perhaps any other, consists in ascribing asthma to a spasmodic or convulsive contraction of the external muscles of respiration, much dependent on habit.

Now, the action of these muscles, so far from being morbid or dependent on habit, is a natural, instinctive and salutary effort to prevent suffocation, the stimulus to which consists in an exaggetion of that which excites the muscles in ordinary respiration—

namely, as above explained, the want of breath, resulting from inadequate oxygenization of the blood. Nothing is more common, for instance, than to see a patient with diseased heart, while sleeping tranquilly, start up and begin to respire with violence. Here it is obvious that the necessity for violent respiration preceded the act; and the necessity depends on impeded transmission of blood through the heart and lungs; for starting is invariably accompanied by palpitation, and preceded by frightful dreams, or some sensation of præcordial distress, indicating an obstructed circulation. I have frequently examined the heart and lungs by auscultation immediately before the supervention of a paroxysm of dyspnœa, and have always found that the heart began either to palpitate, or to act in that irregular, confused, and, as it were, struggling manner, which denotes its engorgement. I was therefore enabled to tell the patient that difficulty of breathing was coming on, to which, with some astonishment, he would reply in the affirmative, being himself forewarned of the approaching accession by a feeling of anxiety and straitness in the præcordia. The fact is so universally true, that any one may satisfy himself of it by entering an hospital and gently placing a patient with orthopnœa from disease of the heart, in a rather uneasy position, when the series of phenomena described will become manifest.

Dr. Burrows communicated to me the particulars of a case, recently under his observation, in which the respiration was alternately violent and tranquil under the following circumstances. The patient dozed for a few minutes at a time, during which his complexion became livid, and his pulse more and more feeble, oppressed and irregular. He then started up, and, after a few violent wheezing respirations, relapsed into the same calm doze. In this case the mitral orifice was contracted to the size of a pea. Now, there can be little doubt that as, during sleep, the stimulus of want of breath is less felt, and the muscles of respiration are, consequently, less excited by it,—in simple language, as the respiration is more feeble during sleep, the lungs were not, in the present case, kept sufficiently expanded to admit of an adequate circulation through them: whence ensued engorgement of the heart and venous system of the body, with insufficient arterialisation of blood in the lungs, and the necessity for breathing resulting from it, which series of phenomena was relieved by the succeeding violent respirations. I have frequently observed this series of phenomena in a greater or less degree: occasionally even in coma. In another case, violent gasping and wheezing respiration, lasting from a few seconds to two or three minutes, occurred at intervals of four or five minutes, during which the patient dozed, even though sitting erect on a stool and undergoing a stethoscopic examination; and this series of actions continued so long as the patient remained disposed to sleep in that situation. In another case of great dilatation and softening, the precise symptoms described by Dr. Burrows occurred for the last week of the patient's life, whether he was awake or asleep, except when calmer sleep was procured by mild opiates. In a third case, a lady had, for several years, observed her husband's respiration, while he was in the horizontal position, but not in the raised position, to be as follows:—after every four or five respirations calmly performed, succeeded a pause of a few seconds; then he started with a "convulsive motion of all his limbs, and a heaving of the shoulders." She had watched this continue for hours together, but he was unconscious of it, and generally slept soundly without frightful dreams. His disease was slight hypertrophy and disease of the aorta.

In all these cases, it is manifest that the action of the muscles of respiration was consecutive to the obstruction of the circulation, and that it was not dependent on any spasm of those muscles, but simply on the necessity for breathing, which instinctively excited them to a salutary preservative effort.

Asthma from disease of the heart often imitates the characters of the other varieties; and this perhaps for a very simple reason, that the lungs are in much the same state as in those varieties. Thus, it is *humid* or *humoral*, when there is permanent engorgement of the lungs, causing copious sero-mucous effusion into the air-passages, as in cases of contraction of the mitral valve. It is *dry*, when the engorgement is only temporary, as in cases of pure hypertrophy. It is *continued*, when there is a permanent obstruction to the circulation; and any of the varieties may be *convulsive*, when the heart has sufficient power to palpitate violently.

The worst cases of convulsive asthma from disease of the heart are those of hypertrophy with dilatation and a valvular or aortic obstruction.

We shall now examine the state of a patient labouring under severe asthma from disease of the heart, and then take a more strictly medical view of the nature and progress of the asthmatic paroxysm.

The respiration, always short, becomes hurried and laborious on the slightest exertion or mental emotion. The effort of ascending a staircase is peculiarly distressing. The patient stops abruptly, grasps at the first object that presents itself, and fixing the upper extremities in order to afford a fulcrum for the muscles of respiration, gasps with an aspect of extreme distress.

Incapable of lying down, he is seen for weeks, and even for months together, either reclining in the semi-erect posture supported by pillows, or sitting with the trunk bent forwards and the elbows or fore-arms resting on the drawn-up knees. The latter position he assumes when attacked by a paroxysm of dyspnœa—sometimes, however, extending the arms against the bed on either side, to afford a firmer fulcrum for the muscles of respiration. With eyes widely expanded and starting, eye-brows raised, nostrils dilated, a ghastly and haggard countenance, and the head thrown back at every inspiration, he casts round a hurried, distracted look of horror, of anguish, and of supplication; now imploring, in plaintive moans, or quick, broken accents, and half-stifled voice, the assistance already often lavished in vain; now upbraiding the impotency of medicine; and now, in an agony of despair, drooping his head on his chest, and muttering a fervent invocation for death to put a period to his sufferings. For a few hours—perhaps only for a few minutes—he tastes an interval of delicious respite, which cheers him with the hope that the worst is over, and that his recovery is at hand. Soon that hope vanishes. From a slumber fraught with the horrors of a hideous dream, he starts up with a wild exclamation that "it is returning." At length, after reiterated recurrences of the same attacks, the muscles of respiration, subdued by efforts of which the instinct of self-preservation alone renders them capable, participate in the general exhaustion, and refuse to perform their function. The patient gasps, sinks, and expires.

Such are the sufferings, in their worst form, of an asthmatic from disease of the heart. We have now to take a more strictly medical view of the nature and progress of the asthmatic paroxysm.

If about to be severe, it is generally preceded by certain premonitory symptoms, which, though not so marked as in ordinary asthma, are much of the same nature—probably because derangement of the circulation and imperfect oxygenization of the blood are present in both. In cardiac asthma, however, many of the nervous symptoms which characterise the ordinary varieties are often deficient. One of the most common and efficient exciting causes of cardiac, as of all other asthmas, is derangement of the stomach, the irritation of which extends to the heart, and stimulates it to inordinate action. The irritation, according to the theory of Sir Charles Bell, or the lately revived excito-motory views of Prochasca, is propagated through the medium of the par vagum, by which nerves the stomach and heart are closely associated. Accordingly, after a feeling of acidity, flatulence, or a load on the stomach from undigested food, often accompanied with abdominal distention, the patient experiences pain, weight, and constriction in the forehead and over the eyes, accompanied (if the case be one of hypertrophy of the left ventricle) with throbbing of the temples and the sound of rushing waters. He feels a sensation, scarcely to be defined, of oppression, tightness and anxiety about the præcordia, frequently with slight palpitation. Sometimes the patient is drowsy, listless, restless, irritable, and impatient, not only of society, but of the attentions of friends: these symptoms, however, are, in general, more prevalent in ordinary asthma. The signs described afford the experienced asthmatic well-known assurance of the approaching attack.

They gradually become worse and worse, especially after a meal, and eventually burst into a paroxysm. The time of the accession is less regular than in ordinary asthma, being more dependent on the state of the heart, which is liable to accidental excitement at any moment, from a variety of causes. The fit, however, as in ordinary asthma, is, on the whole, more apt to supervene during the evening or early part of the night; and this, as appears to me, for two reasons: 1st. the recumbent position is unfavourable to respiration, the diaphragm being pressed upwards by the abdominal viscera, and the expansion of the chest being opposed by its own weight. 2d. During sleep, respiration is not assisted by the will, which, during the wakeful state, from the sensation of want of breath being more acutely felt, is ever ready to maintain the body in the position most favourable to breathing. From the co-operation of these two causes, therefore, the circulation becomes so far embarrassed before the patient is aroused to a sense of his condition, that it can only be relieved by those violent efforts which constitute the asthmatic paroxysm. He accordingly awakes, generally with a start, in a fit of dyspnœa, accompanied either with violent palpitation, or a distressing sense of anxiety in the præcordia and great constriction of the chest, as if it were tightly bound. He is compelled to assume a more erect posture, and intensely desires fresh, cool air; the respiration is wheezing, and performed with violent efforts of all the muscles of respiration, both ordinary and auxiliary. The inspirations are high and accompanied with apparently little descent of the diaphragm, and the expirations are short and imperfect. The surface is chilly, the extremities are cold, and the face is pale and sometimes livid.

In cases in which the pulmonary congestion is only *temporary*, as in hypertrophy either simple or with dilatation, there is no cough beyond a few slight and ineffectual efforts, producing little or no expectoration; and in such cases the fit subsides as soon as the engorgement of the heart and great vessels is relieved, which nature generally effects in two or three hours or less, by determining the blood to the surface and creating diaphoresis. In some instances, I have known this to be regularly accompanied with a copious secretion of pale urine and a purging alvine evacuation (Case of May). In this case, the attacks recurred, according to the assertion of the patient, every night for several years.

The pulse, however full, strong and bounding at first, may, during the worst of the paroxysm, become feeble and small, and the sound and impulse of the heart may be diminished; and this, in cases even of hypertrophy; for the organ, being gorged to excess, is incapable of adequately contracting on its contents.

Such is the nature of an asthmatic fit when the pulmonary congestion is only temporary: the case is different when it is *permanent*, as in valvular disease and in some extreme cases of dilatation. For then, there is violent cough in suffocative paroxysms, accompanied, at first, with difficult and scanty expectoration of viscid mucus, but ending gradually in a copious and free discharge of thin, transparent, frothy fluid, occasionally intermixed with blood. This evacuation, by disgorging the pulmonary capillaries, affords great relief to the cough and dyspnœa. As, however, the transudation of the matter to be expectorated into the air-passages, and its final elimination, are slow processes, paroxysms of this description are much more protracted than those of dry asthma from hypertrophy. They frequently last five or six hours, and I have known them persist, with occasional remissions, for two, three, or more days. During the attack, the pulse is quick, small, and weak, often irregular and intermittent. The slowness which the latter characters sometimes appear to give it, has led some authors to suppose that the circulation through the heart is little disturbed in asthma. This is in some degree true in reference to other varieties of asthma; but it is always incorrect in reference to that from disease of the heart.

As the paroxysm subsides, the anxiety and constriction decrease, the respiration becomes less frequent, high, and laborious, and the pulse becomes slower, fuller, and more regular. But some degree of wheezing and tightness of the chest remain, and the paroxysm is very apt to return for two or three nights successively, and sometimes for a much longer period, until the lungs are freely unloaded by copious expectoration. It may, indeed, continue to recur at brief intervals for an indefinite period, or the patient may never be wholly exempt from some degree of asthmatic dyspnœa.

A severe asthmatic attack from disease of the heart is in general far more injurious in its consequence than one from an affection of the lungs.

SECTION VI.

TREATMENT OF VALVULAR DISEASE.

According to the foregoing principles, (p. 367,) the exciting causes of valvular disease are, 1. over-tension of the valves by the force of the circulation; and 2. inflammation, both acute and chronic.

It has been shown in the chapter on endocarditis, that as it is now possible to detect this disease with much precision, so it is possible, in a considerable proportion of cases, to counteract the establishment of valvular disease by active antiphlogistic and mercurial treatment during the inflammatory periods (p. 219). It has also been shown that acute rheumatism is, of all others, the most frequent cause of endocarditis, and that this frequency may be remarkably diminished by the treatment for acute rheumatism described at p. 179. With respect to valvular diseases resulting from causes other than inflammation, it is almost impossible to obviate their formation, since there are no positive signs of the latent mischief but what result from the disease already formed—from the obstruction itself. As, in the present state of our knowledge, we are not acquainted with any means of *removing* a valvular disease once established, whatever be its cause, the indications of treatment in such cases are, to prevent its increase, to counteract its tendency to induce hypertrophy and dilatation, and to relieve the symptoms of an obstructed circulation. The extreme importance of obviating the supervention of hypertrophy or dilatation, has been explained at p. 371 et seq.

The remedies calculated to answer these indications, are, in general terms, such as diminish the force and activity of the circulation: namely, occasional venesection to a moderate extent, in certain cases; an unstimulating and rather spare, though sufficiently nutritious diet; a tranquil life, with respect both to the body and the mind; and a good state of the digestive organs and alimentary canal.

The extent to which any remedy must be carried, can only be determined by the particular circumstances of each case. If, for instance, the patient be robust and plethoric, depletory measures may be pursued to a greater extent, and *vice versâ*. In general, if the valvular obstruction is not very considerable, and there is no hypertrophy or dilatation, and no tendency to plethora, an abstemious light diet, comprising a moderate proportion of animal food, and a scrupulously tranquil life, with a regular state of the bowels, constitute all the prophylactic treatment that is necessary; and it is satisfactory to know that, by these means, danger may in many instances be completely averted. I have several times known patients with a moderate—even with a rather considerable valvular obstruction, attain the age of sixty, seventy, and even eighty, though the symptoms, judging from their account, had commenced in early life.

On the other hand, if precautionary measures be neglected and hypertrophy or dilatation superinduced, there is no organic disease of the heart, except adhesion of the pericardium, which tends more rapidly to its fatal termination. Hence the great importance of detecting and attending to disease of the valves in its earliest stage.

When the obstruction is very considerable, has produced hypertrophy or dilatation, and is attended with much dyspnœa, orthopnœa, and dropsy, the case is one of the most difficult that the practitioner can encounter. The most urgent symptoms, however, generally admit of being removed for a time; and the amelioration which takes place is sometimes truly astonishing. But, unhappily, the complaint seldom fails to return with greater or less promptitude. If the patient be youthful and of a robust constitution, the relapse may not occur for several months, especially if he have not been affected with dropsy, or only for the first time; but if he be of a shattered constitution, and have previously had severe attacks, the symptoms commonly return the moment he resumes any active occupations. In an ulterior degree of the disease, no sooner are the symptoms dispersed than they return, though the patient have not been guilty of any indiscretion. When this is the case, the fatal event is never far remote, and may be expected to occur at any moment.

The remedies suitable for the treatment of the cases described, are, abstractions of blood, purgatives and hydragogues, diuretics, sedatives, revulsives, a well-regulated, moderate, unstimulating diet, and, what is paramount in importance to all, complete repose. These remedies, however, are not to be employed at random: so used, they might not only be unavailing, but directly destructive. It is only by adapting them to the character of the organic cause of the disease, and to the constitutional condition, —only, in short, by a sound diagnosis, that they can be administered safely and effectually. It is necessary, therefore, to enter into further particulars relative to their nature and mode of application, and this may be most conveniently done by adverting separately to each.

Blood-letting.—When, with the valvular obstruction, there is hypertrophy or hypertrophy with dilatation, bleeding is generally necessary, and may be repeated, in small quantities of four to six ounces, two, three, or more times, according to the strength of the patient and the urgency of the palpitation and dyspnœa. It should not, however, be employed if the patient be anæmic, or on the verge of that state. It should also be avoided, if possible, in the aged. Some authors, as Laennec, have recommended that blood-letting be practised in valvular disease in the unsparing manner of Albertini and Valsalva. The results of my own experience lead me to dissent entirely from this doctrine. Excessive bleeding cannot remove the valvular obstruction—cannot, therefore, *cure* the disease; consequently its employment with this object is inappropriate. It is, moreover, directly injurious; as it reduces the patient to a state of anæmic debility, which increases his palpitation, renders his circulation more liable to be embarrassed by the valvular obstruction, and greatly augments the disposition to general dropsy. I have always observed blood-letting to be most serviceable in valvular disease when carried only just so far as to relieve the existing urgent symptoms without encroaching on the constitutional powers.

If, instead of hypertrophy, dilatation, either simple or attenuated, be conjoined with valvular disease, blood-letting is less necessary, and is more injurious if carried to excess. It should be resorted to reluctantly; only when imperiously demanded by excessive dyspnœa, which other means have failed to relieve; the least quantity that suffices to afford relief should be drawn; and the depletion should not be repeated if it can possibly be avoided. Attention to these rules is still more necessary in the aged. The greater the valvular obstruction, the greater is likely to be the embarrassment of the circulation, with its train of formidable symptoms, if the power of the heart and the system be reduced below a certain point. Of this I feel satisfied from reiterated observations.

Diuretics.—When there is dropsy and a scanty secretion of high-coloured urine, remedies of this class are of the greatest utility. In most cases, indeed, the dyspnœa, palpitation, cough, &c. decrease in the same proportion as the urine increases and the dropsy disappears. Nor is it only when dropsy has actually appeared that diuretics are useful. They are remarkably beneficial in any stage of the disease; for, by drawing off the serous portion of the blood, they diminish the quantity, without deteriorating the quality of that fluid, and thus relieve palpitation and dyspnœa, and obviate infiltration, without materially reducing the strength.

Diuretics are very variable in their effect, a weaker sometimes answering perfectly after a stronger has failed. When, therefore, one does not speedily produce the effect, another should be tried. The surest way is to employ several at once. A pill consisting of three grains of blue pill, one of pulv. scillæ, and one or half of one of pulv. digitalis, given three or four times a day, seldom fails: or it may be given once or twice a day with a draught of Tr. scillæ. mxx. Sp. ætheris. nit. and Sp. Junip. C. comp. āā. ʒss. ad ʒi, in Dec. Spartii. ℥iss. twice or thrice a day. I have sometimes found all these fail until ʒij or ʒiij of infusion of digitalis was added to the draught. Its effect, however, must be carefully watched. Bitartrate of potass is always a valuable auxiliary, and may be given to the extent of ʒij or ʒiij in twenty-four hours, either in the form of a drink, of electuary with honey, or suspended in the above draughts. Some writers strongly recommend doses of ℥ss. twice or thrice a day, and I have seen them produce surprising effects on dropsy, but some care is requisite to obviate hypercatharsis. The acetate and hydriodate of potass and Tinct. Lyttæ are also valuable diuretics. In old or feeble subjects, a vehicle of Inf. Gentianæ or Cascaril. is useful as a tonic.

In very feeble and reduced patients, dropsy should not be too rapidly evacuated; as the process is attended with a degree of exhaustion which is often fatal. The period, indeed, immediately succeeding the disappearance of dropsy is, on this account, one of the most critical. The older physicians were aware of this, and ascribed it to the accumulation of fluid in the internal cavities. Such, however, is not always the cause; for, in cases that terminated fatally at the period alluded to, I have frequently ascertained, both by auscultation, percussion, and post-mortem examination, that the internal and external dropsy disappeared simultaneously. The exhaustion alluded to should be obviated by strong beef-tea, or animal food if it can be digested, and, if necessary, by stimulants, of which Sp. armorac. C. or gin punch, being diuretic, are the best.

Purgatives.—When diuretics fail to remove dropsy, purgatives will frequently produce that effect. The two classes of remedies may, indeed, be combined with great advantage, when the patient is strong enough to bear them. The drastic hydragogue purgatives are the most efficacious, as tinct. jalapæ, elaterium, &c. The effects of the latter are sometimes truly astonishing. I have seen an extreme, universal anasarca removed by it in three or four days. The remedy is apt, however, to be excessively violent in its operation, and it should, therefore, be given to strong subjects alone; or the weakly and aged should be carefully watched. As its effect varies in different individuals, it should be tried at first in small doses, as from one eighth to one fourth of a grain. With caution it may be carried to two grains. I generally give it in the form of pills with pulv. capsici, which obviates its griping effect; sometimes I add a grain or two of calomel, which prevents vomiting. A single pill should produce six or eight watery evacuations, and it may be given two or three mornings successively; or every second or third morning, according to the strength of the patient. If much exhausted, he may take gin punch more freely. All the other purgatives may be useful, especially such as produce watery evacuations. A very good one is, the infusion of senna, with tinct. jalapæ ʒi, and tartrat. or acetat. potass ʒij. to iv. I have several times known both diuretics and hydragogues signally fail till the patient was put upon a dry diet.

An occasional purgative is sometimes very beneficial though there be no dropsy; as, for instance, when an asthmatic attack has appeared to be induced by an excess of bile, by undigested food, or by acrid or long-detained fæces in the intestines. Under such circumstances, a purgative often alleviates, and sometimes terminates the attack. Except with a view of removing dropsy, or plethora in cases where hypertrophy is conjoined with valvular disease, frequent, systematic purging should be avoided on the same principle as blood-letting: viz. lest it should too much reduce the system and occasion anæmia.

Diaphoretics.—When there is anasarca, cutaneous transpiration contributes very powerfully to remove it. A lady under my care, and subject to frequent attacks of anasarca, often found the swelling disappear in twenty-four hours with copious perspiration. Strong, stimulating sudorifics, however, should be avoided, as they are both too debilitating and too exciting to the circulation. Gentle saline diaphoretics are the best, and their effect may be promoted by warm clothing, and the occasional use of the warm bath to keep the skin soft and open. When there is no anasarca, and no *permanent* pulmonary engorgement with expectoration, diaphoretics, beyond warm clothing, are of little use, except occasionally, to relieve asthmatic attacks. For the latter purpose I have generally found them of great utility; but, as internal remedies of this class are slow in their operation, they should be assisted by fomenting the hands and feet, or immersing them in warm water, at the same time keeping the trunk covered. If perspiration can thus be gently elicited without heating and stimulating the patient, it is one of the most effectual means of curtailing a paroxysm. Nature herself indicates the remedy, as an asthmatic paroxysm often terminates with spontaneous diaphoresis. In one patient under my observation (May) this occurred nightly, and to an extreme degree, for several years.

Emetics.—These are extremely useful, or extremely pernicious, according as they are judiciously given, or the reverse; and it is only by a sound diagnosis that the practitioner is enabled to judge whether they can be safely administered or not. When there is an undigested, bilious or acid load on the stomach, exciting a fit of palpitation or asthma, its removal by an emetic often affords instantaneous relief. But the medicine should be one which simply evacuates the stomach without much shaking the system, as ipecacuan, with sulphate of copper or of zinc, but by no means potassio-tartrate of antimony.

If the disease of the heart and the embarrassment of the circulation be great, even such an emetic cannot be given without danger of aggravating all the symptoms. I have seen emetics, administered under these circumstances, exasperate and prolong the paroxysm, increase the frequency of its recurrence, and speedily bring the patient to his grave. They may even cause death during the paroxysm. Their dangerous effect consists in their increasing the engorgement of the heart and the obstruction of the circulation. For this reason, they should not be ventured upon in disease of the heart, simply for the object of promoting expectoration—an object which may by other means be much more safely and effectually accomplished. In other varieties of asthma, on the contrary, especially that from pituitary catarrh, they are peculiarly beneficial by promoting the expectoration of the immense accumulations which take place in the lungs. Hence the importance of carefully distinguishing between these two classes of cases.

I have said thus much respecting emetics, because they have been alternately both extolled and decried, the parties using them under different circumstances, and neither perfectly understanding on what their good or bad effect depended.

Though emetics are objectionable except for the purpose of evacuating the stomach, small doses of ipecacuan or tartrate of antimony are useful as diaphoretics and expectorants. When the obstruction of the circulation is great, they cannot safely be carried to nausea, as this state is apt to bring on a languor of the circulation which leads to the formation of polypi in the heart. In the case of a lady lately under my care, nausea came on unexpectedly, and independent of tartar emetic, at the moment when she had just been relieved of an excessive dropsy: it was followed by suffocating dyspnœa, an imperceptible pulse, and other symptoms indicating the formation of a polypus in the heart. She died in a week, and the polypus was found.

Puncturing.—When dropsy has failed to be relieved by other means, and the cutaneous tension has become intolerable, the practitioner is compelled to resort to puncturing. I say compelled, because the remedy is a last and dangerous resource. The danger, however, may be considerably diminished by making 20 to 30 small punctures with a grooved needle in the thighs and trunk, but never below the knee, and allowing the fluid to ooze *slowly* during four or five days or a week. When *incisions* are made with a lancet or scalpel, especially below the knee, and the fluid is evacuated quickly, as in twelve to forty hours, the patient, according to my observation, generally dies. This event sometimes results from sloughing of the incisions, but more commonly from exhaustion induced by the *sudden* evacuation of the fluid. In one instance I saw the patient die from hæmorrhage.

Setons, issues, and blisters on the præcordial region, are of no use unless there be chronic inflammation of the heart: in other cases, the pain and irritation that they occasion are often injurious.

Expectorants.—When there is permanent engorgement of the lungs, free expectoration always affords relief, and I have seen great dyspnœa result from its suppression by an incipient catarrh, a dry sharp atmosphere, and even a dose of laudanum. Many asthmatic fits dependent on valvular obstruction terminate with copious expectoration of thin sero-mucous fluid. This secretion, therefore, should always be maintained when there is a tendency to it.

As the stomach in disease of the heart is extremely fastidious and delicate, oily, sweet, and nauseous expectorants should be carefully avoided. Squill with an acid, as the acetic or nitric, has been found by experience to be the most efficacious remedy of this class. Vinegar of squill has been highly extolled by Floyer, and Tinct. Scillæ, g^{tt} x. Acid. Nitrici, g^{tt} vi. Extr. Hyoscyami, gr. iij. and Aquæ puræ, ℥iss, as a draught every three or four hours during the paroxysm, is the favourite prescription of Dr. Bree for the asthmatic paroxysm of his *first species*, i. e. "from effused serum in the lungs." Mist. ammoniaci, though in general too heating for the young, is a useful expectorant for the old, when sufficiently diluted. The same may be said of the decoction of seneka. Ipecacuan and tartrate of antimony, in small doses, are valuable expectorants as well as diaphoretics. They may be carried to a slight degree of nausea, if the obstruction of the circulation is not very great. Phlegm accumulates during sleep, and it is for this reason principally, that the patient suffers more on first rising in the morning. The elimination of the phlegm is greatly facilitated by a cup of any hot fluid, especially coffee; and, to allay the nervous irritability of the lungs which generally leads to coughing before the phlegm is sufficiently detached to be thrown off with ease, I have found from half a drachm to a drachm of tinct. camphoræ comp. of great utility.

Expectorants should not be constantly given, but only to relieve an asthmatic paroxysm, or to restore the pulmonary secretion when accidentally suppressed.

Gases.—The effects of atmosphere on asthmatics are so diversified that they can scarcely be reduced to any general rule. When, however, expectoration is habitually copious, a moist warm atmosphere favours it, probably by relaxing the pulmonary vessels. A clear, sharp air, on the contrary, checks it and thus increases dyspnœa. Again, such an air relieves dyspnœa when it depends, not on engorgement of the lungs, but on a languid action of the heart, as in dilatation with attenuation; and this it does by stimulating and bracing the system, and causing a freer circulation through the lungs and more perfect arterialization of the blood. Electricity appears to act in the same way when it produces any good effect. I have never tried the inhalation of oxygen, but it is highly commended by Dr. Beddoes and others; and it is rational to think that, in suffocative dyspnœa from retardation of the blood in the lungs, it would relieve the anxiety and straitness by causing a more perfect arterialization.

Smoking tobacco or stramonium sometimes affords extraordinary relief to asthmatics, and this it does partly, perhaps, by increasing the bronchial and salivary secretion, but more especially by its sedative and antispasmodic effect in tranquillizing the nervous system, resolving the bronchial spasm, and allaying the sensation of want of breath. The experience of the patient is the only certain criterion of its utility. In many cases I have certainly seen it prejudicial. Its utility is the greatest in those who are of a highly nervous, irritable habit, and in whom asthma displays most of the spasmodic character.

Antispasmodics.—While the Cullenian doctrine, that spasmodic constriction of the bronchi was the sole cause of asthma, prevailed, remedies of this class were much in vogue; but experience has not realised the high expectations to which the theory gave rise,—a result which is not surprising, since it has been shown that there is almost always an organic cause in addition to the bronchial spasm. Antispasmodics are useful auxiliaries, but cannot be depended upon alone. When they contribute to diffuse and equalise the circulation in disease of the heart, they are beneficial: when they fail to produce this effect, they are of little use. In an incipient paroxysm from slight disease of the heart, I have frequently found a draught of sp. ammoniæ aromat. or fœtid. with æther and laudanum, promptly restore the colour to the face, and warmth with perspiration to the skin, with general relief. In one case of hypertrophy with dilatation, adhesion of the pericardium, and aortic regurgitation, a glass of gin and water had always the effect. Sometimes gr. x to xv of carbonate of ammonia is more efficacious than any other remedy. The solution of assafœtida has also appeared to me to be very powerful, but few patients can be prevailed upon to take it.

In most instances, the antispasmodic, whatever it be, is productive of eructation, and to this, in some measure, I attribute its beneficial effect, as flatulence alone suffices to occasion a paroxysm. The eructation sometimes occasioned by the remedies themselves, especially æther, must not be mistaken for the extrication of real flatus.

When the paroxysm is fully established, and results from a great degree of organic disease of the heart, antispasmodics have little or no effect in affording relief; and large doses of sedatives, as opium, hyoscyamus or conium, or of stimulants as æther, often prolong it. In conjunction with other means, however, moderate doses may be tried, and, if the patient feel himself relieved, they may be continued, and *vice versâ.*

Digitalis, according to my experience, is an excellent adjunct to an antispasmodic draught: gtt xx or xxx of the tincture may be given every three or four hours, with gtt vi to x of tinct. opii, or, if that disagree, of hyoscyamus, in cinnamon water. Care should be taken to intermit the digitalis before its specific poisonous effect is produced.

In suffocative, agonizing orthopnœa, when the restlessness and jactitation of the patient aggravates the distress, I have often found narcotics afford great relief simply by inducing sleep and a diminished sensation of suffering, and they should always, I think, be used under these circumstances, to procure the patient a remission when the fatal event is close impending. The doses should be small; as, in this oppressed state of the brain, average doses are very apt to occasion narcotism.

Stomachics.—The correction of dyspepsia is of the first importance in organic disease of the heart; as palpitation is often dependent upon it alone. Two gentlemen at present under my care for hypertrophy with dilatation, never suffer palpitation, dyspnœa, or headache, except when affected with acidity, flatulence, &c. Such cases are often mistaken for "*the stomach*" alone;—a most dangerous mistake. Of the individuals alluded to, one has had a fit of apoplexy, and the other has been repeatedly rescued from it by prompt cupping. When there is acidity, antacids, of which chalk is the most certain, should be freely given every third or fourth hour, its constipating effect being counteracted by the previous or simultaneous exhibition of a few grains of rhubarb. I have already stated that the stomach, if loaded, should, in the first instance, be evacuated by a gentle emetic; copious draughts of tepid water or chamomile tea being taken to ensure its full and easy effect. This treatment will generally terminate an attack dependent on dyspepsia, in two or three days, and sometimes in as many hours. Towards the close of the attack, sedatives, as opium or hyoscyamus, assist by tranquillizing the nervous system.

Not only antacids, but also acids themselves, have been proved by experience to correct acrimony of the stomach accompanied with flatulence and distention. Their efficacy is the greatest when the acrimony is bilious, and they then act, in all probability, both by neutralizing the alkaline qualities of the bile, and exciting the stomach to an altered and more healthy secretion. That they possess the latter property, is to be inferred from their correcting acidity and preventing fermentation even when there is no bile. A sour apple is a popular remedy for heart-burn. The acids to be employed, are, the mineral acids much diluted, and also the acetous. Saccharine acids, as oxymel, acescent fruits, raspberry vinegar, &c. should be avoided, as they are apt to be more injurious by their fermentation, than beneficial by their acid qualities. Acids need not be tried till antacids have failed, which is seldom the case.

To give tone to the stomach, bitters are very useful. Infusions should be employed during the paroxysm, as tinctures are too stimulating; but after the second or third day, when the patient begins to amend, either the one or the other may be used. The bitters may be conjoined with the antacids, &c.

Tonics.—When disease of the heart is of the hypertrophic kind with increased activity of the circulation, tonics are obviously inappropriate: when it is of the dilated kind, with languor of the circulation and atony of the system, they are remedies of the greatest value, and it is mainly by them that a complete cure can be effected. All the tonics may be used according to the discretion of the practitioner. In pale, anæmic subjects, the preparations of iron, in full doses, for one or two months, are by far the best; and there is none preferable to the mist. Ferri Comp. Of the advantages of bracing air and exercise and of the shower bath, I have spoken in the article on dilatation. A discreet use of the cold bath also, is highly beneficial.

Diet.—When valvular disease is complicated with hypertrophy and increased activity of the circulation, animal food should be only sparingly allowed, as on alternate days; but a full proportion should be restored whenever pallor, weakness and increased palpitation indicate the supervention of anæmia. When dilatation or softening attends valvular disease and causes feebleness of the circulation, the diet should comprise a full, or even a large, proportion of animal food, provided the digestion will bear it. In all circumstances of valvular disease, the diet should be plain and regular, dyspeptic articles should be excluded, and the individual meals should be moderate in quantity.

Such are the remedies to be used in the treatment of organic disease of the heart. It cannot be too strongly inculcated on the practitioner, that the disease, when remediable, is not to be cured by *relieving* the paroxysm, but by *preventing* its occurrence. Every attack gives the patient much ground to retrace: a single attack may undo the progress of a year, and death may result from the indiscretion of a day. Great firmness is necessary on the part of the physician to impress this strongly on the mind of the patient; for the latter, when his *feelings* are easy, can seldom—very seldom, be made to comprehend that the necessity for his rigid adherence to medical, regiminal, and dietetic discipline, is equally imperative.

The practitioner, however, is not the less to study the means of relieving the paroxysm; not only because, in it, he has perhaps the greatest of human sufferings to alleviate, but because, by curtailing the attack, he increases the chances of a cure.

CHAPTER X.

ANEURISM OF THE AORTA.

SECTION I.

CLASSIFICATION, NOMENCLATURE, ANATOMICAL CHARACTERS AND FORMATION OF ANEURISMS OF THE AORTA.

Aneurism ('Ανεύρυσμα, τος, τὸ, arteriæ dilatatio et inde ortus tumor, from 'Ανευρύνω, dilato, amplio) is an enlargement of a portion, or the whole, of the circumference of an artery.

Aneurisms of the aorta are divided by authors into four species.

1. *Dilatation*, which is an enlargement of the whole circumference of the artery.

2. *True aneurism*, which is a sacculated dilatation of a portion only of the circumference, or of one side of the artery.

3. *False aneurism*, which is formed by ulceration or rupture of the internal and middle coats, and expansion of the external or cellular into a sac. It is called *primitive* when all the coats are divided, as by a wound; and *consecutive* when it is consequent on ulceration or rupture of the internal and middle coats.

4. *Mixed aneurism*, which is a supervention of false upon true aneurism, or upon dilatation: that is, after dilatation either partial or general of all the three coats, the internal and middle burst, and the external alone expands into a further sac, surmounting the original dilatation or true aneurism.

1. *Dilatation, or Enlargement of the whole circumference of the Aorta.*

When the coats of the aorta, whether from inflammation or from any other morbid action, have become diseased, they lose their elasticity, a quality which resides principally in the middle tunic. As fluids press equally in every direction, the blood propelled by each contraction of the heart into the aorta, exerts not only a longitudinal, but a lateral force, which expands the vessel, and constantly tends to enlarge its calibre. The elasticity of the arterial walls, in the healthy state, enables the vessel to resist this expansive force, and to regain its previous calibre after the diastole. Consequently, when the elasticity is impaired or lost by disease, the vessel, not being able to regain its original dimensions after each diastole, becomes permanently dilated, and this takes place to a greater or less extent, and with greater or less promptitude, in direct proportion to the predominance of the distending over the resisting force.

It very rarely happens that a dilated aorta does not present, in its interior, some of the morbid changes already described: (see Arteritis, p. 222:) namely, cartilaginous, steatomatous, atheromatous, or calcareous depositions, with a thickened, wrinkled, and fragile state of the internal coat. When such depositions are not apparent, the walls, according to my observation, are always more or less indurated, opake and inelastic; and also sometimes extenuated, particularly the middle coat, and sometimes thickened, with a softened and easily separable state of the internal coat:—conditions which are a much more natural cause of dilatation than paralysis of the middle coat, supposed by some authors to be its cause when no depositions are manifest.

The ascending portion and arch of the aorta, particularly the latter, are by far the most frequent seats of dilatation,—probably because they are, from vicinity, most exposed to the expansive force of the left ventricle; but the descending portion, both in the chest and abdomen, is sometimes affected, and the dilatation is then either uniform throughout the whole length of the vessel, or it consists of one, or even a series, of ovoid or fusiform expansions. The side of the artery adherent to the spine, and the lesser curvature of the arch, yield less readily than the other parts. Dilatation of the aorta does not in general exceed twice the natural calibre of the vessel, but I have occasionally seen it attain three, and even four times that size. When such is the case, it frequently presents many minor bulgings or pouches, which give it a considerable resemblance to the transverse arch of the colon. The walls of these pouches are often extenuated and semi-transparent from horn-like and calcareous depositions, and it is here more especially that mixed aneurism is apt to take place; for the brittleness of the depositions causes rupture of the internal and middle coats, and the engraftment of false aneurism upon the true.

Dilatation of the pulmonary artery is extremely rare. I have met with one remarkable case in which it was enlarged to four inches and a half in circumference (Wetherall), and another in which it was rigidly ossified, even beyond its primary subdivisions in the lungs (Lady R.).

Dilatations, even though pouched, scarcely ever contain laminated coagula; for the surface is in general too smooth to arrest the blood: when they do take place, it is in consequence of an ulcerated or fissured state of the internal membrane which forms nuclei for the adhesion of fibrine.* The coagula thus formed occasionally fill up the whole of the dilated portion, and leave the canal of the artery of its natural calibre.

The great arterial trunks rising at right angles from the aorta, as the innominata, left carotid, and cœliac, generally participate in the dilatation: the left subclavian almost always remains exempt; without doubt, says Laennec, on account of the acute angle at which it branches off.

Dilatation takes place not only in the aorta and its immediate trunks, but sometimes in smaller and more remote arteries, as, for example, the carotid by the side of the sella turcica, and the arteries of the circle of Willis, of which I have seen several instances; the temporal,† cœliac, mesenteric, and emulgent,‡ with their ramifications, the arteries of the extremities, and those feeding tumours of any description, particularly *fungus* hæmatodes and the hæmorrhagic nœvus or aneurism by anastomosis of John Bell.

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* Case by Burns, on Disease of the Heart, p. 206; and by Bertin and Bouillaud, Obs. xxxvi.

† Cruveillier Essai sur l'Anat. Patholog., Paris, 1816, tom. ii. p. 60.

‡ Journal de Méd. par M. M. Corvisart, Leroux et Boyer, tom. vii. p. 255.

2. *True Aneurism, or lateral, partial Dilatation of the Aorta.*

True aneurism differs from dilatation in the circumstances, that it is an enlargement of a limited portion only of the circumference of the aorta; that it generally rises with an abrupt margin; and that its neck is, in most cases, narrower than the body of the sac (Case of Hill). Its formation is to be attributed to a loss of elasticity and resistance in the particular part only that dilates; and the proofs of its existence, in contradistinction to false aneurism, consist in the possibility of tracing the internal and middle coats of the artery throughout the whole extent of the expansion, and in the presence, within the sac, of those morbid appearances, which are peculiar to the internal coats of arteries; such as calcareous, cartilaginous and atheromatous depositions, slight fissures and small red spots. These proofs have of late years been so frequently verified by dissection, that the reality of aneurism by dilatation of all the coats of an artery is no longer problematical.

Almost all the aneurisms of the ascending portion and arch are originally of the true species, but the false is sometimes engrafted upon them. The tumour generally springs from the anterior, or the lateral parts of the vessel, while the posterior part and the lesser curvature of the arch are little, if at all implicated:* it sometimes attains the magnitude of a mature fœtal head,† and almost invariably inclines to the right side of the chest, except when it originates beyond the middle of the arch. When it springs from the root of the aorta, and the middle and internal coats happen to burst, there results, not a false aneurism surmounting the true, as in other parts, but a fatal extravasation into the pericardium. The reason of this is, that the part of the aorta referred to, is destitute of the cellular tunic, and the pericardium which supplies its place, not being equally extensible, bursts, rather than dilates into a false aneurism. In the same way, the deficiency of the cellular coat in the arteries of the brain, causes their rupture to be followed by an apoplectic extravasation, instead of by the formation of a false aneurismal sac: of true aneurisms, however, I have met with two instances.

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* An aneurism, however, of the abdominal aorta, a little above the origin of the cœliac artery, sprung from the posterior side of the vessel, in a case by Dr. Beatty, Dub. Hosp. Rep. vol. v. p. 188.

† Corvisart, Journ. de Méd. par M. M. Corvisart, Leroux et Boyer, tom. vii. p. 355. Laennec de l'Auscult. tom. ii. p. 691.

It has been stated by a recent writer that a preparation in Mr. Hunter's collection subverts the doctrine that "false aneurism does not form at the root of the aorta." The preparation of which he speaks, however, scarcely subverts this doctrine, since it is not one of *false* aneurism; for the middle coat is perfect, the internal one alone being either diseased, or removed, (which, is doubtful,) at the base of the sac. Though it has been denied by authors that *false* aneurism may form at the root of the aorta, it has not been denied that *true* may. I have myself seen it in more than one instance (e. g. case of Mitchell). Coagula are occasionally, but not often, found in true aneurisms; they are usually in masses, adherent by peduncles, and seldom in layers investing the walls, unless the aneurism be very large: the reason of which is, that, the mouth of the sac being in general spacious, the blood has a sufficiently free ingress and egress to circulate with force, while the surface of the sac is so smooth as not to arrest the fibrine and cause its deposition in layers. But when the circulation is by any cause enfeebled, the blood stagnates and forms coagula in masses, which become adherent by limited portions or peduncles. True aneurism is much more rare than either false, mixed, or dilatation.

3. *False Aneurism, or Aneurism by Ulceration of the Internal and Middle Coats.*

Nichols proved, by experiments made before the Royal Society of London, that when the internal and middle coats of an artery are divided, and water or air forced into the vessel, the external coat is distended so as to form a small sac (Philosoph. Trans. vol. xxxv. p. 443). In the same manner, when the internal and middle coats are perforated by ulceration or a fissure, the blood, by its lateral pressure, gradually raises the external coat and expands it into a sac, which communicates by a narrow aperture or neck with the interior of the artery, whose calibre is not enlarged. As the distention proceeds, the external coat itself gives way, and the sheath of the vessel next opposes the effusion of blood: finally, when this also yields, the contiguous parts, whatever be their texture, contribute to the formation of the sac, they having previously undergone thickening and agglutination by chronic adhesive inflammation, to which distention or pressure had given rise.

Such is the manner in which the sac is formed in aneurism from ulceration of the arterial coats. It presents no vestige of the middle or fibrous coat, nor the depositions connected with the cellular tissue of the internal membrane; but its inner surface is extremely rugged and unequal from lymph irregularly deposited by inflammation. To this rugged surface adhere the layers of fibrine subsequently separated from the blood.

Perforation of the internal and middle coats is not always followed by aneurism of the kind described. Laennec met with a case in which the internal and middle coat had been divided by a narrow transverse fissure extending over two-thirds of the circumference of the artery, and the blood, instead of distending the external coat into a sac, had insinuated itself between it and the fibrous, and dissected them from each other round upwards of half the circumference of the artery, from the arch of the aorta down to the common iliacs.* Fissures of the kind described, result from cracks or lacerations following the circular direction of the fibres of the middle coat, or from lesions occasioned by calcareous depositions; but the case of Laennec, and two similar ones mentioned by Mr. Guthrie,† are the only instances within my knowledge in which a fissure has been followed by more than a circumscribed effusion of blood around it, occasioning a slight swelling of the external coat. Nichols found this in the body of George the Second,‡ and Hodgson once met with it.§

The late Mr. Shekelton has described, in the Dublin Hospital Reports, third volume, another, and previously unnoticed kind of aneurism: the blood had forced its way through the internal and middle coats, dissected the middle from the external or cellular for the space of four inches, and then burst again through the internal and middle coats into the canal of the artery, thus forming a new channel, which eventually superseded the old one, the latter having become obliterated by the pressure of the tumor.

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* De l'Auscult. tom. ii. p. 700.

† Guthrie on the Diseases of Arteries, pp. 40 and 43.

‡ Philos. Trans. vol. lii. p. 269.

§ On Diseases of Arteries, p. 63.

The causes of perforation of the internal and middle coats and the formation of false aneurism, are, 1st. *ulceration*, generally occasioned by the detachment of calcareous incrustations, by atheromatous depositions under the internal membrane, and, more rarely, by tubercles, or small abscesses in the substance of the fibrous tunic: 2d. *rupture or cracking*, which takes place when the tunics have been deprived of their elasticity by cartilaginous, steatomatous, fungous and calcareous degeneration.[*] The immediate or exciting cause of the rupture is generally some violent exertion or accident; and in most instances patients with aneurism date it from some occurrence of this kind. Rupture does not appear ever to take place in a perfectly sound artery; and, if it did, the experiments of Dr. Jones seem to prove that it would not be followed by an aneurism, as an effusion of lymph takes place, which strengthens the vessel in the lacerated part.[†]

While aneurisms of the ascending aorta and arch are, in the first instance, almost invariably true, though they occasionally become mixed; those of the descending aorta are generally false;[‡] and the calibre of the artery is, with few exceptions, not in the slightest degree dilated opposite to the tumor.

Aneurism by perforation of the internal and middle tunics, is the only species of which Scarpa admits the reality: but the inaccuracy of his opinions has been fully proved, and, as before stated, there is no longer any question respecting the actual existence of aneurism by dilatation of all the coats.

The cases of false aneurism that are on record, are very numerous. Reference may be made to the works of Lancisi, Morgagni, Guattoni, Scarpa, Desault, Warner, Hodgson, Horne, Laennec, Bertin, and Bouillaud.

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[*] Scarpa on Aneurism, § 20, 21, 22. Laennec de l'Auscult. tom. ii. p. 704. Hodgson, p. 62.

[†] Jones on Hæmorrhage, p. 125.

[‡] I lately met with a mixed one.

4. *Mixed Aneurism or False Aneurism surmounting True.*

This species is formed in the following manner. All the three tunics of the artery first undergo an expansion, which, according to its form, constitutes either a dilatation, or a true aneurism: as the expansion proceeds, the internal and middle tunics burst, and the external, being more extensible, dilates into a sac, surmounting the original enlargement.

Aneurisms of this description are very numerous.

The true and the mixed varieties of aneurism communicate with the cavity of the aorta by an aperture more contracted than the body of the tumor, and presenting a prominent border. This disposition of parts has been perfectly described by Scarpa, and admirably represented in his plates.

General Observations on Aneurism of the Aorta.—Haller, and M. M. Dubois and Dupuytren have remarked a variety of aneurism, in which the internal membrane makes a hernia through the ruptured fibrous coat and lines the sac, which is formed by the external or cellular coat. Hernia of the internal membrane may occur, according to Laennec, in very small aneurisms: he had seen it in two, which were not larger than cherries; but, when the tumor increases, the internal membrane speedily bursts. This he found to have been the case in two other aneurisms which did not exceed the size of walnuts (De l'Auscult. ii. p. 693). The experiments of Mr. Hunter, Scarpa, and Sir E. Home prove, that when the external and middle coats of an artery are removed, the internal one does not dilate into an aneurism, but either bursts, or is strengthened by granulations arising from its surface, and by adhesions formed with the surrounding parts.

Corvisart having found several firm, solid tumors of the size of nuts, intimately adherent to the aorta, while the external and middle coats appeared to be deficient at the point of attachment, was led to imagine that *extraneous* tumors, for such he conceived them to be, becoming adherent to arteries, led to the formation of aneurism.* Hodgson, on the contrary, regards the tumors in question as instances of aneurism cured, the sac having been filled up by lamellated coagula,* and the volume of the tumor diminished by absorption; and Laennec, Bertin, and the best authorities subcribe to his opinion.

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* Essai sur les Maladies du Cœur, p. 313.

As an aneurismal sac enlarges, the surrounding parts become involved in its composition. Thus, the bones, muscles and various other structures, often contribute to its formation. The viscera, also, become implicated when the tumor is situated in their vicinity; and the membranes with which they are invested, being distended to their utmost, finally yield, and the sac bursts into their cavities. Accordingly, aneurisms frequently prove fatal by discharging their contents into the lungs, æsophagus, stomach, intestines, bladder, &c.

The size which the tumor attains depends upon the nature of the surrounding parts, and is very much determined by their extensibility—a property which is almost in direct proportion to the quantity of cellular tissue of which they are composed. Hence it is, that, when the disease is situated at the root of the aorta, where the pericardium supplies the place of the more extensible cellular coat of the vessel, the sac bursts into the pericardium before it has attained any great magnitude. Hence, also, it is, that in the cranium, where the arteries are destitute of the cellular coat, and are ill supported by the pia mater and the soft pulpy substance of the brain, aneurism is extremely rare; for such a lesion of the coats of the arteries as would elsewhere give rise to aneurism, is here attended with rupture and apoplectic effusion. It has been already stated, however, that the arteries of the brain are not unsusceptible of dilatation and true aneurism.

One of the first circumstances that almost invariably follows the formation of false aneurism, is, the deposition of the fibrine of the blood upon the internal surface of the sac. This deposition takes place in successive concentric layers, which have a different aspect according to the date of their formation. The most central consist simply of blood more or less firmly coagulated, and they are probably formed subsequent to death: a little farther, the coagulum is dryer, paler, and evidently composed of a larger proportion of fibrine, with less serum and colouring matter: still farther, are layers of pure, whitish, yellowish, or greyish fibrine; and finally, in contact with the walls of the cyst, are layers of the same matter, but completely opake, of a somewhat friable consistence like dryish paste, and very closely resembling flesh which has been deprived of its colour by boiling. The most recent layers adhere to each other so slightly as almost to float within the sac; those beneath are united by a downy or villous cellular tissue, the adhesion being stronger in proportion as the layers are older. Patches of vivid red formed by reticulated blood-vessels, are occasionally found in the fibrine, and blood often penetrates between its layers, and stains those which are friable, or decomposed. Coagula are softer in some cases than in others, though the physical circumstances be the same in both. The difference is probably owing to a difference in the chemical constitution of the blood, some specimens containing a larger proportion, and more healthy quality, of fibrine.

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* On Diseases of Arteries, p. 127.

From these anatomical characters, it is evident that lamellated coagula form by successive depositions of the fibrine of the blood; and the depositions are accounted for by the stagnation of the blood within the sac; for it is proved by experiment and observation that coagulation of this liquid takes place whenever its course is interrupted; hence the polypi that are found in the heart, the great veins, and the arteries, when the circulation through these parts is obstructed.

The coagulation of blood within a false aneurism is favoured by two circumstances—the narrowness of the aperture of communication with the artery, and the ruggedness of the interior of the sac. In true aneurism, as before stated, the width of the aperture of communication and the smoothness of the interior of the sac, are unfavourable to the coagulation, and accordingly fibrinous *layers* are very seldom found in those aneurisms unless they be of great size, although they often contain coagula in masses attached at one part only, by a peduncle of greater or less thickness.

The thickness of fibrinous depositions within aneurisms is sometimes very great. Most commonly, it is from half an inch to an inch and a half, but I have seen it exceed three inches. The thickness is generally greater in one part of the sac than in another. Laennec has seen fibrinous coagula as compact and diaphanous, as horn softened to the utmost by heat, and of a thickness exceeding five fingers' breadth.

Aneurisms, and the diseases of the coats of arteries which precede their formation, are much more frequent in men than in women. Of sixty-three cases seen by Hodgson, fifty-six occurred in the former and only seven in the latter (On Diseases of Arteries, p. 87). I have found the proportion in females rather larger than this, with respect to aneurism of the aorta; but, with respect to external aneurism, it is much smaller, perhaps not exceeding one in fifteen to twenty. The causes of disease of the arterial coats leading to aneurism, have been fully discussed in the chapter on arteritis (p. 226).

SECTION II.

PATHOLOGICAL EFFECTS OF ANEURISMS OF THE AORTA ON CONTIGUOUS PARTS.

The pathological effects of aneurisms of the aorta on contiguous parts, vary according to the volume, the form, and the position of the tumor.

Dilatation, when not very considerable, produces little derangement of the surrounding parts. For, as the swelling is equable and diffuse, it does not exert a pressure on any one organ in particular, and its magnitude is not such as to create much inconvenience from general infarction. The worst of its effects are those which it produces on the trachea and great bronchi; for, though the pressure be slight, it often suffices, in consequence of the great irritability of these parts, to occasion considerable dyspnœa. It must not, however, be imagined that dilatation is an unimportant affection. It will hereafter be shown that when complicated with enlargement of the heart, which it generally brings on, it is one of the most formidable diseases incident to the circulatory apparatus.

An aneurism which forms a defined tumor, whether it be of the true, or the false species; whether it be large, or small, may produce the most pernicious effect. These are, 1st. Such as result from compression of the neighbouring parts.

2nd. Such as result from their destruction.

1st. By compression, the functions of the lungs, bronchi, heart and œsophagus, are deranged, and that sometimes to a fatal extent. In the abdomen the functional derangements are comparatively inconsiderable, and very rarely endanger life. The reason of this is two-fold; first, that the abdominal organs are not of so vital a nature as the thoracic; and secondly, that the tumor, instead of being pent up in a rigid, bony case like the chest, is permitted, by the yielding of the intestines and the distensibility of the abdominal parietes, to expand freely in almost every direction. Pressure on any particular organ, therefore, is in a great measure obviated by the want of counter-pressure or a fulcrum. A ventral aneurism, however, even though not much larger than an egg, when seated behind the stomach, I have known to produce severe and obstinate symptoms of dyspepsia; as anorexia, nausea, flatulence, acidity, insatiable craving, occasional pains in the epigastric and hypochondriac regions shooting through to the spine, constipation, and progressive emaciation. A case of this kind was under my care last summer. Dr. Graves relates a similar one in the Med. Gaz. vol. xx. p. 66. Ventral aneurism, also, sometimes deranges the respiration by preventing the due descent of the diaphragm—an effect which may proceed either from the magnitude alone of the tumor, or, what is much more common, from its being seated near, or in the substance of the muscle, and impeding its motions. Ventral aneurism is also occasionally attended with involuntary evacuation of the urine and fœces, by remarkable alternations of constipation and diarrhœa, and by deep-seated excruciating pains, resembling those of lumbar abscess. These symptoms arise from compression and irritation of the cœliac, hypogastric, and other plexus of organic nerves. A deeply interesting case illustrative of this, has been published by Dr. Beatty in the Dubl. Hosp. Rep. vol. v. p. 166.

2nd. The consequences of destruction of contiguous parts are far more formidable than those resulting from their compression.

When the tumor exerts an unusual pressure on any organ or texture, adhesive inflammation takes place and unites the parts in contact. As the pressure increases, absorption and, ultimately, perforation of the sac ensues, causing death by internal hæmorrhage as the immediate consequence. The perforation takes place either by sloughing or by laceration, according to the nature of the membrane or texture perforated. Thus, when the tumor advances to the skin, or when it extends into a cavity lined by a *mucous* membrane, it bursts by the separation of a slough which has formed upon its most distended parts, and not by laceration. On the contrary, when the sac projects into a cavity lined by a *serous* membrane, sloughing of the membrane does not take place, but the parietes of the tumor, having become extremely thin in consequence of distention, at length burst by a crack or fissure, through which the blood is discharged. An aneurism may burst into a great variety of parts, which we shall notice in succession.

When the lungs are in contact with the tumor, adhesion, absorption of the sac, and rupture of the pleura take place, and the effused blood deluges the bronchi and causes suffocation (Case of Lafin). I have known two other instances of this.

It often happens that an aneurism of the ascending aorta, or arch, compressing the trachea or one of the great bronchial trunks, opens its way into it by ulceration of the cartilaginous rings and sloughing of the mucous membrane, and causes suddenly fatal hæmoptysis.

More rarely, perforation takes place into the œsophagus, and death then ensues from hæmetemesis.

Aneurisms occasionally burst at the origin of the aorta, and cause death by effusion of blood into the pericardium. The fatal event, however, is not always so sudden as in the preceding cases; a circumstance which Laennec attributes to the pericardium being supported, and the effusion consequently restrained, by the general infarction of the chest resulting from the presence of the tumor. This reason appears to me unsatisfactory, because, as before explained, aneurisms at the root of the aorta generally burst before they attain any considerable magnitude: nor, if large, would the resistance offered by the atmospheric pressure in the lungs equal the force with which the blood tends to escape into the pericardium—a force equal to the propulsive power of the left ventricle. It is, perhaps, more probable that the inextensibility of the pericardium beyond a certain point, and the resistance of the heart to compression, form the principal powers which limit the effusion of blood. It would appear that life is sometimes protracted for a considerable period after the rupture of the sac; for, in specimens presented to the Société de la Faculté de Médecine by M. Marjolin, the margins of the aperture, according to Laennec, were polished, as if of old standing and, as it were, fistulous.* Rupture into the pericardium is very rare. Laennec never met with an instance. The first that has fallen under my own observation, occurred in 1830, at St. George's Hospital. Morgagni† and Scarpa,‡ however, have collected together a considerable number of these cases, and Hodgson saw two, the aneurism beginning half an inch above the semilunar valves, and occupying the whole ascending aorta and arch.

I have met with one instance of an aneurism at the origin of the aorta, bursting into the right ventricle (Case of Mitchell).

Aneurisms have been known, though very rarely, to burst into the pulmonary artery. M. M. Payen and Zeink saw an instance,§ and Dr. Wills another.‖ My friend Professor Monro showed me a preparation of an aneurismal pouch springing from the aorta directly against the pulmonary artery; and it is probable that, if the patient's life had been prolonged, rupture would have taken place into the artery. His son, Dr. David Monro, favoured me with the case of Evans, in which were two openings out of the aorta into the pulmonary artery.

The left cavity of the pleura and the posterior mediastinum are the parts into which thoracic aneurisms most frequently burst. It is extremely seldom, on the contrary, that they open into the right pleura.

Laennec has seen an aneurism of the descending aorta, which had compressed and destroyed the thoracic duct, and produced engorgement of all the lacteal vessels.

Aneurisms sometimes compress the descending vena cava, and cause cerebral congestion, œdematous intumescence of the face, and even apoplexy. I have met with several instances of this kind.

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* Laennec Op. Cit. ii. p. 715.

† Epist. xxvi. Nos. 7, 17, 21.

‡ On Aneurism, § xix. p. 103 et sequent.

§ Bulletin de la Faculté de Médecine, 1819, No. 3.

‖ Trans. of Soc. for the Improvement of Med. Chirurg. Knowledge, vol. iii. p. 85.

Corvisart,[*] and Bertin and Bouillaud[†] each cite a case of apoplexy thus occasioned. I have repeatedly noticed varicose enlargement of the veins on the sternum and upper ribs, resulting from the same cause, and also great venous intumescence round the root of the neck.

Another effect of aneurisms is, to obliterate arteries springing from them, or contiguous to them. I have met with two cases in which both the left carotid and subclavian were plugged up at their origin from the tumor.[‡] The obliteration is sometimes effected, not by a plug of lymph, but by contortion or compression of the vessel. Mere contraction of the origins of arteries from these causes is very common.

Ventral aneurisms may open their way into the various abdominal viscera, as the intestines, the bladder, &c., as well as into the cavity of the peritoneum. In a case by Dr. Beatty, and another by Dr. Stokes, they burst into the left cavity of the pleura.

It is also stated that they may burst under the peritoneum, and that the patient may survive for months or even years, while a succession of pulsating tumors are gradually formed in the left hypochondriac, lumbar, iliac, and inguinal regions, which diminish the impulse and murmur of the original aneurism (Dr. Cowan's Manual, p. 43).

Aneurisms not only cause destruction of the soft parts; but, what is still more remarkable, erosion of the bones. This phenomenon has been variously explained. The old pathologists erroneously ascribed it to a chemical solvent power of the blood. Hunter, Scarpa, and Hodgson thought that it resulted from absorption of the earthy matter, in consequence of the pressure of the sac. Corvisart and Laennec attribute it to a sort of detrition or wearing down, produced by a purely mechanical action. Bertin and Bouillaud believe that it is more or less dependent on inflammation. To myself it appears that absorption and mechanical detrition are the principal agents concerned in producing the effect. That pressure is capable of exciting absorption of bone, is certain, as the vertebræ have been found excavated by an aneurismal tumor without being divested of their periosteum;* and there can scarcely be a doubt that, when a denuded bone is exposed to the constant dashing of a column of blood, it undergoes disintegration by the mechanical detachment of its particles.

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[*] Journal de Médecine, par M. M. Corvisart, Leroux et Boyer, tom. xii. p. 159.

[†] Traité des Maladies du Cœur, p. 137.

[‡] Cases of Aneurism, vii. viii. Lond. Med. Gaz. Sept. 12, 1829, p. 449.

Whether inflammation ever contributes to the effect, is difficult positively to determine. Analogy does not discountenance this opinion, yet appearances are adverse to it; as pus has never been found on bone eroded by an aneurism; as exfoliation scarcely ever takes place, and as nothing is discovered on it analogous to the cicatrization or irregular reproduction observable in other bones when affected with caries.

Cartilage, whether exposed to the action of the blood in aneurismal sacs, or to the pressure alone of the tumor, either remains entirely uninjured, or suffers incomparably less than bone. This is most manifest in the intervertebral substance and the cartilages of the false ribs. The circumstance is attributable to the elasticity of cartilage, which protects it from mechanical disintegration, and to its less highly organized structure, which renders it little susceptible of absorption, or ulceration. The bones liable from their position to be eroded by aneurism are, the vertebræ, the sternum, the ribs, and sometimes the ossa ilii.

It is principally by aneurisms of the descending aorta that the vertebræ are injured. In these cases, the portion of the sac in contact with the vertebræ is entirely destroyed, and its borders adhere very firmly round the eroded part of the bone, on which the blood plays freely in consequence of the fibrinous layers having been absorbed at that part. The destruction is sometimes so deep, that the shell of the vertebræ forms the only partition between the sac and the spinal canal. Very rarely, however, does rupture take place into the canal. I am not aware that there are more than three instances on record; one by Laennec, in the Revue Médicale for 1825; another, of which the preparation, by Mr. Chandler, is in the Hunterian Museum; and a third by Dr. Beatty, in the Dub. Hosp. Rep., vol. v. p. 188.

Ventral aneurisms seldom produce this effect, because the abdominal viscera and walls yield to the tumor. When, however, the tumor springs from the posterior side of the aorta, and is braced down by the crura of the diaphragm, as in Dr. Beatty's case above quoted, erosion may take place: I have also seen a tumor so braced down by the pancreas, right kidney, and pyloric end of the stomach,—all cemented together by old adhesions, that, if the patient had survived sufficiently long, I should think that spinal erosion would have taken place.

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* Hodgson, p. 79.

When the spinal nerves are irritated by erosion, the usual neuralgic and paraplegic symptoms may be experienced in the lower extremities, as in Dr. Beatty's case.

It is by aneurisms of the ascending aorta and arch that the sternum and ribs are eroded. The tumor generally presents on the right side, if it spring from the ascending portion of the vessel; but if it affect the centre of the arch or the innominata, it usually projects at the upper part of the sternum and about the sternal ends of the clavicles, which have even been dislocated from this cause. When the tumor is connected with the posterior or descending part of the arch, it shows itself underneath the left clavicle.

According to Hodgson, when the periosteum contributes to the formation of the sac, its vessels continue to secrete an earthy matter, which, in some instances, has been deposited to such an extent as to form a considerable portion of the tumor.

Small aneurisms have the effect of destroying bones in a greater degree than large: a circumstance attributable to the greater concentration of the pressure exercised by them.

SECTION III.

SIGNS AND DIAGNOSIS OF ANEURISM OF THE AORTA.

In the present section, the general and physical signs will be described separately: in the next, a brief synopsis will be given of the two conjointly, with reference to the several forms of aneurism.

General Signs of Aneurism of the Aorta.

When an aneurism is buried deep in the chest, and not capable of being detected by the sight and touch, it does not present a single general sign which is peculiar to itself, and therefore pathognomonic of its existence. There are even cases in which it occasions no functional derangement—no inconvenience whatever; and the first circumstance that unveils the truth, is, the sudden death of the patient while apparently in the enjoyment of perfect health. I have met with six or seven instances in which large aneurisms have existed without awakening even a suspicion in the minds of the medical attendant. One, in particular, eluded the penetration of a distinguished foreign auscultator, though he explored the lungs with eminent success.

There is only one general sign of aneurism of the thoracic aorta which is unequivocal and certain: namely, a tumor presenting externally, and offering an expansive as well as heaving pulsation, synchronous with the action of the heart.

Of the remaining general signs, a large class are identical with those of organic disease of the heart: viz. palpitation, dyspnœa, cough, tendency to syncope, terrific dreams, starting from sleep, hæmoptysis, livid or otherwise discoloured complexion, cerebral or hepatic congestions, serous infiltration, &c. This identity arises from an identity of cause; namely, an obstacle to the circulation, which depends either upon the aneurism alone, or conjointly upon it and a disease of the heart, to which, sooner or later, the aneurism almost invariably gives birth, if seated in the ascending aorta or arch, but very rarely, if seated beyond those parts. I have already shown (p. 301) that when the aneurism is unconnected with hypertrophy or dilatation of the heart, it may subsist for a long period, even for years, without producing any material symptoms of an obstructed circulation: consequently, under such circumstances, the symptoms above enumerated will be slight or wholly absent. But when the aneurism becomes complicated with disease of the muscular substance of the heart, marked obstruction of the circulation ensues, and the above symptoms may attain the highest degree of intensity. From the whole argument, therefore, it results, that as the symptoms in question are slight when resulting from aneurism alone, and only considerable when it is complicated with disease of the ventricles, they are but equivocal and unsatisfactory signs of the aneurism in particular.

There are, however, certain other general signs which are more characteristic: yet even these are ambiguous and unsatisfactory; as they only bespeak lesions of the viscera, or derangement of their functions, but do not proclaim the latent cause of the mischief. But when they coincide with the signs derived from auscultation, they lose their ambiguity and rise into real importance; for the two classes of signs, general and stethoscopic, are a commentary on each other, and reciprocally borrow a precision and certainty of which they are individually destitute.

I shall succinctly describe the general signs to which I refer, and subjoin to each the principal sources of fallacy. The means of detecting the latter, I shall point out in the final synopsis.

1. When the tumor has attained a considerable magnitude, the cavity of the chest is preternaturally filled, and the patient complains of a sense of constriction, infarction, and oppression.

But these sensations are common to almost all diseases of the chest.

2. The radial pulses are sometimes dissimilar, or one is extinct —an effect dependent on obstruction, or obliteration, of the arteria innominata, or left subclavian.

But the difference of the two pulses at the wrist may proceed from a variety of causes independent of aneurism of the aorta, as contraction of the origin of either subclavian from osseous, cartilaginous, steatomatous, or other depositions; obstructions in the course of the artery, occasioned by tumors, wounds, aneurism, &c.; an irregular subdivision of the humeral, brachial, or radial artery. I have known the most ludicrous surmises occasioned by the radial crossing to the outside at the middle of the fore-arm, and the superficialis volæ supplying its place at the wrist.

3. When the origin of either subclavian is contracted, the pulse at the corresponding wrist is later than the ventricular systole in a greater degree than natural,—for, in the healthy state, it is a little later.

I have not found this symptom uniformly present under the circumstances in question. Besides, the heart is more frequently its source than the aorta, and I have observed it to be most considerable in cases of regurgitation into the left auricle; but obstruction of the aortic valves may occasion it in a minor degree, particularly if this lesion be accompanied with attenuation or atony of the ventricular parietes. Even dilatation with attenuation, softening, and, in short, any cause weakening the expulsion of the blood out of the left ventricle, may give rise to it. When the sign exists in both pulses, the presumption is strong that its source is in the heart.

4. According to Corvisart, a purring tremor, the *frémissement cataire* of Laennec, is sometimes perceptible to the hand at the middle or upper part of the sternum, and indicates aneurism of the ascending aorta.

Purring tremor *above* the clavicles is an almost constant concomitant, and therefore a valuable sign, of dilatation of the arch; but, according to my experience, it is unfrequently and imperfectly occasioned in that situation by sacculated aneurisms, especially if lined by strata of lymph. I have never known the tremor to be occasioned below the clavicles by dilatation, unless the enlargement was so great as to extend beyond the lateral margins of the sternum, and allow the tremor to be felt through the intercostal spaces: but I have met with one case in which a dilatation of the pulmonary artery, though not voluminous, afforded a marked tremor between the cartilages of the second and third ribs on the left side: this, however, is not remarkable, as the artery, about an inch and a half above its origin, *naturally* lies nearly opposite to the part described, when the patient is in the horizontal position. I have never known a sacculated aneurism create a tremor below the clavicles, unless the tumor had eroded the bones of the chest and presented externally, underneath the integuments; yet I can believe that there may be cases, though I do not happen to recollect one, in which the tremor is perceptible through the costal interspaces.

But the purring tremor may be occasioned in any part of the chest by mucous rattles, particularly those of the snoring kind, in the large bronchial tubes; and I have observed that, when derived from this source, it is a very common cause of deception with young auscultators, in reference both to aneurisms of the aorta, and valvular diseases of the heart. The fallacy may be effectually avoided by simply requesting the patient to hold his breath.

5. When the trachea, or primary bronchial divisions are compressed by an aneurismal tumor, a harsh wheezing or sibilous sound, proceeding deep from the throat, characterizes the respiration; the voice is either croaking, or reduced to a whisper, or it is a compound of both; respiration may be feeble in one lung, and puerile in the other, from compression of one of the primary bronchi; the breathing is often extremely laborious, and, when the heart is simultaneously diseased, asthmatic dyspnœa sometimes occurs in paroxysms of the most suffocating severity. When the œsophagus is compressed, deglutition of solids is rendered difficult, and sometimes impracticable; for the descent of the morsel excites an excruciating pain from the summit of the sternum to the spine, or lancinating deeply in every direction through the chest.

But compression of the trachea, or œsophagus, with the above symptoms, may be occasioned by tumors of any description,—even by aneurism of the innominata (Case by Dr. Stokes, Dub. Jour. v. p. 406). Wheezing respiration may proceed from an accumulation of glutinous mucus in the great bronchi, and it is common in all forms of asthma. I have likewise known it produced in an extreme degree by chronic laryngitis with thickening of the soft parts covering the arytænoid cartilages, and also by ossification and ulceration of the larynx from strumous, syphilitic, and mercurial disease. I have also known it produced by chronic, strumous hypertrophy of the tonsils, which in one instance occasioned suffocation. So difficult was it, before the discovery of auscultation, to distinguish the seat of wheezing respiration, that it has in many instances been imputed to an affection of the larynx, when it was, in reality, occasioned by an aneurism of the aorta, and, under these circumstances, bronchotomy has several times been actually performed with the view of obviating suffocation by the supposed laryngeal affection.

6. When the vertebræ are eroded, the patient suffers an intense terebrating pain in the spine; and when the brachial plexus of nerves is compressed by the tumor, an aching sensation pervades the left shoulder, neck, scapula and arm, with numbness, formication, and impaired motive power of the limb.

But I have met with cases in which nearly similar pains were experienced, although there was no destruction of the vertebræ; and it is common to hear individuals affected with rheumatism or spinal disease make the same complaints. I have several times met with intense neuralgia of the neck, shoulder, and arm, from malaria, and yielding to quina and iron. Aching pain down the inside of the arm may also be occasioned by various forms of organic disease of the heart, and it thus constitutes a part of that concatenation of symptoms which is denominated angina pectoris. I have likewise often met with it in hysterical females subject to palpitation, in nervous males, in very plethoric individuals, and occasionally in cases of pericarditis. In all these cases, the pain probably originates in irritation of the cardiac plexus of the pneumogastric, propagated to the internal cutaneous nerves.

7. When, in consequence of an adhesion between the aneurismal sac and the pleura, the blood plays upon the lungs, a sense of ebullition is said to be experienced.

But the same symptom is familiar to individuals labouring under phthisis, or chronic mucous catarrh; and it proceeds from the successive bursting of large bubbles, formed by the transmission of air through the fluid in tuberculous caverns, or in the greater bronchial ramifications.

8. It occasionally happens that the patient suffers excruciating pain from a spasm, pursuing the course of the diaphragm, and binding the chest around, as with a cord.

This symptom is too vague to be important, and it also occurs in hysteria, gastrodynia, colic, spinal diseases, and rheumatism of the diaphragm.

9. A pulsation felt underneath the sternum or ribs at the superior part of the chest.

This, although one of the least equivocal signs of aneurism, is not without ambiguity. It may be occasioned by a tumor of any description, as an enlarged gland, or a cancer, interposed between the sternum and the aorta, and receiving the pulsation of the latter.

10. A pulsation is felt above the sternum or clavicles.

But this may be occasioned, 1. by enlarged glands or other tumors seated on the subclavian artery, and receiving its pulsation. 2. By varix of the jugular vein about its junction with the subclavian. In five cases, I have seen immense swelling of this kind occasioned by encephaloid tumors of the right lung compressing the descending cava. Both of the preceding conditions have deceived expert practitioners. 3. By subclavian aneurism. This affection sometimes resembles aneurism of the aorta so exactly, that it is extremely difficult to distinguish them. Allen Burns records a case in which all the eminent surgeons of the district were unanimous in pronouncing the affection subclavian aneurism; yet it proved to be aortic.* Sir A. Cooper has published a number of similar cases; and one is mentioned by Professor Monro tertius.† 4. By aneurism of the arteria innominata or the carotid. In April, 1826, I saw a case at Guy's Hospital, which led to much deliberation respecting the propriety of taking up the carotid above a pulsating tumor, supposed to be an aneurism of that artery. It was finally decided that the tumor was too low, and the design was judiciously abandoned. The affection proved to be a dilatation of the aorta and arteria innominata. The carotid was sound. This state of parts was indicated to me by the stethoscope. Mr. Hodgson met with a similar case.‡

11. The superior and middle parts of the chest are dull on percussion. But this sign I have not found to occur, unless the aneurism was larger than an egg; and, moreover, it is common to several other diseases, as encephaloid tumors of the lungs and anterior mediastinum; hydropericardium, which, if very great, may mount as high as the second rib; pleuritic effusion, which, if confined to one side, may mount almost to the clavicle; circumscribed empyema at the upper part of the chest,—of each of which diseases I have seen repeated instances.

12. An enlarged and varicose state of the subcutaneous veins over the upper part of the chest, especially the sternum.

But this may be occasioned by any tumor compressing the descending cava within the chest, of which I have seen five instances from encephaloid disease of the right lung: it may also be occasioned in a less degree by any disease of the heart which occasions a great impediment to the circulation through the right side of the organ.

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* Surg. Anat. of Head and Neck, p. 30. † Elements of Anat., vol. ii. p. 249.

‡ On the Diseases of Arteries, p. 90.

It cannot be a subject of surprise, that a series of symptoms liable to so many fallacies should have proved insufficient, without the aid of auscultation, to dissipate the deep obscurity which involved the diagnosis of aneurisms of the aorta.

Physical Signs of Aneurism of the Aorta.—The investigations of M. Laennec on aneurism of the thoracic aorta were limited and inconclusive. Accordingly, he remarks that, "Of all the severe lesions of the thoracic organs, three alone remain without pathognomonic signs to a practitioner expert in auscultation and percussion—namely, aneurism of the aorta, pericarditis, and concretions of blood in the heart previous to death."

I shall first present the opinions of Laennec, respecting the physical signs of aneurism of the aorta, and then offer the results of my own researches, by which I hope to make it apparent that this malady is characterized by sufficiently pathognomonic signs.

Laennec's opinions are as follows:—On applying the cylinder, in two instances, to tumors presenting externally, he found that their pulsations were exactly isochronous with the pulse; that the shock and sound greatly exceeded those of the ventricles; that the beating was distinctly audible on the back, and that the second sound could not be distinguished at all. For the last reason he denominated the aneurismal pulsation *simple*, in contradistinction to that of the heart, which has a double sound. From these two cases he felt certain that, in some instances, pectoral aneurisms might be recognized by the *simple pulsation*, usually much stronger, both in impulse and sound, than that of the heart; but he thought that, in a larger proportion of cases, the sign would be insufficient: for, as the slightest dilatation of the heart renders its sounds audible over the whole sternum, and even below and along the clavicles, he imagined that, under such circumstances, the first or systolic sound of the organ would be confounded with the sound of the aneurism, with which it is synchronous; while the second or diastolic sound, being audible as far as the tumor, would lead the auscultator to suppose that he there heard the beating of the heart, and not that of the aneurism. I shall presently show that this reasoning is incorrect.

As the second sound is not audible over the abdomen, Laennec found no difficulty in recognizing ventral aneurisms by the *simple pulsation*.

According to my experience, the cylinder is scarcely less capable of affording decisive indications of pectoral, than of ventral aneurism. It is unimportant whether the pulsations be "*simple*" or "*double*," for, though double, they may be distinguished from the beating of the heart, by unequivocal criteria: viz.

1st. The first aneurismal sound, coinciding with the pulse, is *different* from the first sound of the heart: it is a murmur,—varying, indeed, in its pitch, and softer or rougher, according to the circumstances of each case, but still a murmur; and it is to this murmur that the loudness of the sound is attributable, when it exceeds that of the ventricular systole.

2. Supposing that there is no valvular disease of the heart, the aneurismal murmur, explored from its source in the direction of the apex of the heart, becomes progressively weaker, till, on arriving at a point about an inch above the apex, it is wholly inaudible or very feeble and remote, while the first sound of the heart itself, a totally different sound, is louder at this spot than at any other. It is impossible, therefore, to confound an aneurismal murmur with the first sound of the heart in its healthy state. But supposing that it is not healthy: supposing that it has been converted into a murmur by valvular disease; that murmur, as it attends the first sound, will necessarily be seated either in the auricular valves from regurgitation, or in the semilunar valves, from an obstruction in them or the orifice: in which cases the diagnosis will be as follows:—

a. In the case of auricular regurgitation: If a murmur be heard distinct and loud at the upper part of the aneurismal tumor—the part most remote from the apex of the heart; or, in fact, at any part above the third rib, the auscultator may rest assured that, in the absence of semilunar murmurs, it proceeds from an aneurism, rather than from the auricular valves; as murmurs of the latter are always either very weak, or wholly inaudible, so far off. On the other hand, he may be assured that there is simultaneously an auricular regurgitant murmur, if he find it loud and near-sounding about an inch above the apex of the heart, where the aneurismal murmur, in its turn, is very weak or inaudible. The principle of this diagnosis is precisely the same as of that by which semilunar, are distinguished from auricular murmurs (see p. 90).

b. If the valvular murmur be seated in the semilunar valves, the diagnosis is somewhat more difficult, though in general practicable. It has already been shown, (p. 90,) that a murmur of these valves is propagated two inches or more along the course of the vessel, whether the aorta or pulmonary artery, in which it originates; and it has also been shown, (p. 384,) that when the murmur is *considerably* louder and on a higher key two inches or more up the vessel, especially the aorta, than opposite to the valves, it results from roughened coats, or dilatation, or aneurism of the vessel. When the aneurism forms a tumor at the side of the sternum, if the murmur is loud on the outer or humeral side of the tumor, it may be pretty confidently referred to an aneurism; since a murmur of the semilunar valves is very feeble or wholly inaudible so far from the course of the great vessels. If an impulse on the tumor accompany such a murmur, the evidence of aneurism is almost positive.

3. As it has been shown in the preceding paragraphs that the first sound of an aneurism can be distinguished from the first sound of the heart, whether natural or with murmur, the presence or absence of the second sound of the heart on the aneurismal tumor is unimportant; yet even this sound can generally be traced, with a progressive increase of intensity, either to its immediate source, the semilunar valves, or to the line of the aorta and pulmonary artery, along which and the sternum it is propagated as far as the clavicles. The second sound of an aneurism is occasionally attended with a feeble murmur, arising from the expulsion of a portion of its blood by the elastic contraction of its walls during the ventricular diastole. This effect would be greater during an unfilled state of the arteries, as from general anæmia or aortic regurgitation,—a state in which, as shown by Dr. Corrigan, there is a greater flux of blood into and out of aneurisms.

Such an aneurismal murmur with the second sound, is easily distinguishable from the murmur of semilunar regurgitation, by the latter being audible down the course of the ventricles, and by its being exceedingly prolonged; namely, through the whole diastole and period of repose: neither of which circumstances obtains in the aneurismal murmur.

This murmur, again, cannot be confounded with an auricular murmur attending the second sound, because the latter, when it exists at all, (which I find to be exceedingly seldom,) is always too feeble to be audible in the situation of an aneurism.*

A few remarks may now be made on the character of the aneurismal murmur with the first sound; as it is in general rather peculiar, and therefore distinctive. It is a deep, hoarse tone, of short duration, with an abrupt commencement and termination, and often, but by no means invariably, louder than the most considerable murmurs of the heart. It accurately resembles the rasping of a sounding-board, heard from a distance; whereas the sound occasioned by valvular disease of the heart is less hollow and more prolonged, with a gradual swell and fall. The depth and hollowness of the tone is generally greater above the clavicles than below; which is probably attributable to its being reverberated through the chest before it arrives at the ear. This probability is countenanced by the following considerations: *a.* That, in several cases with which I have met,† while the sound, above the right clavicle, was loud and hoarse, it was merely a whizzing without hoarseness, on the superior part of the sternum, where the dilated ascending aorta was in apposition with the bone, and where, consequently, the sound was transmitted immediately to the ear. *b.* That, in the heart, when we listen to its sounds directly through the solid parts where they are uncovered by lung, morbid murmurs are less hoarse and hollow than those occasioned by pectoral aneurisms. *c.* That, in aneurisms of the abdomen and extremities, where there is little or no reverberation of sounds, there is a still less degree of hoarseness and loudness.

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* In the first edition of this work, some obscurity crept into the diagnosis of the aneurismal sounds, in consequence of its being then unknown that the closure of the semilunar valves was the cause of the second sound. Since this was demonstrated by my experiments, (p. 25 et seq.,) the author of the *Rational Exposition* (now called *The Pathology* and *Diagnosis of Diseases of the Chest*) has made several unsound criticisms on my original mode of diagnosis, but he has not had the ingenuity to supply its defects.

† See, for instance, cases of aneurism of the aorta, Lond. Med. Gaz., Sept. 12, 1829, case 9.

The abruptness of the aneurismal sound, compared with the prolonged, swelling character of ventricular murmurs, is owing to the latter being generated by a gradual muscular contraction, or a prolonged semilunar regurgitation, while the former is due to the sudden propulsion of a fluid through a vessel naturally very resistant, and rendered still more unyielding by disease; or through an abrupt orifice into a sac, which enjoys little latitude of motion.

The loudest aneurismal sound is that occasioned by dilatation: and it has more of the grating or rasping character, in proportion as the interior of the vessel is more overspread with hard, and especially osseous asperities. When the dilatation is confined to the ascending aorta, the sound, impulse, and purring tremor above the clavicles, are stronger on the right, than on the left side; and the sound along the mesial part of the sternum—the tract of the ascending aorta, is often superficial, and of a whizzing character.

Old aneurisms, the parietes of which are thickened by fibrinous depositions, yield only a dull and remote sound. In all cases of dilatation, and in the majority of sacculated aneurisms, the sound is loudest above the clavicles, even though the impulse be stronger below. In some cases of the sacculated species, it is louder on the side of the neck opposite to that where the tumor exists. I have found this to proceed from one or other of two causes—first, disease of the inner coat of the aorta before or beyond the tumor, and opposite to that side of the neck where the sound was loudest; secondly, the interposition of the sac, thickened with fibrinous layers, between the aorta and the superclavicular region, in consequence of which the source of sound,—the mouth and cavity of the sac, was unusually remote on the side occupied by the tumor. In one case, where the aneurismal murmur was barely audible, I found the tumor interposed between the sternum and the origin of the aorta, pushing the latter upwards of three inches back. The weakness of the sound was therefore owing partly, no doubt, to the remoteness of the aperture into the sac, but partly also to the inexpansibility of the tumor itself, occasioned by its osseous case in front.

The sound of aneurisms is in most instances audible on the back; and when the tumor occupies the descending aorta, and is extended along the spine, it is often louder behind than on the breast. If it possesses, on the back, the abrupt, rasping character, the evidence which it affords is almost positive; for the loudest sounds of the heart, when heard on the back, are so softened and subdued by distance, as totally to lose their harshness.

Dr. Corrigan has shown, as already stated, that, *cæteris paribus*, the murmur and tremor of an aneurism are stronger during the lax state of the arteries resulting from anæmia or aortic regurgitation; because there is a greater flux of blood into and out of the aneurism, and greater latitude for vibration both in the fluid and the walls of the sac, than when the vessels and the tumor are completely and tightly distended.

Purring tremor is another characteristic of the aneurismal pulsation. It is more considerable in simple dilatation than in sacculated aneurism, particularly if the former be accompanied with much asperity of the internal membrane. From numerous dissections, the fact appears to me to admit of the following explanation:—in cases of dilatation, the interior of the vessel is almost invariably rugged from steatomatous, osseous, cartilaginous, or other adventitious depositions; and the blood, in permeating such a tube, necessarily occasions a strong tremor, as its particles are thrown into preternatural commotion and collision, not only by the enlargement of the calibre of the vessel at the dilated part, by which they are diverted from their direct course, but also by the roughness of the surface of the vessel, by which they are reflected in endless conflicting currents from its sides. In sacculated aneurism, on the contrary, though a portion of blood descends into the sac, and may there create a tremor if the amount and velocity of the current be sufficient; yet the greater quantity pursues a direct and tranquil course along the smooth canal of the artery; and the tremor is therefore less considerable. It is rarely occasioned at all by old aneurisms; because, in consequence of their magnitude and the thickening of their sacs with fibrinous coagula, they possess little susceptibility of vibration.

Purring tremor proceeding from organic disease of the aorta, may easily be distinguished from that occasioned by anæmia.

The former is constant even during a tranquil state of the pulse; it is restricted to a limited space above the sternal extremities of the clavicles, and is accompanied with the hoarse aneurismal sound. Anæmic purring tremor, on the other hand, is only occasional, occurring when there is palpitation from nervous or physical excitement; it extensively pervades the adjoining arteries, the concomitant sound is comparatively soft and feeble, and it is always attended with the venous murmur in the jugulars.

Pulsation attends every species of enlargement of the aorta. In dilatation, it exists above the sternal ends of the clavicles only, and always on both sides of the neck simultaneously; though, when the enlargement is confined to the ascending aorta, it is stronger on the right than on the left side. When dilatation is of a pouched form, and of great magnitude, it may occasion pulsation under the sternum. Of this I have met with instances. Carotid and subclavian aneurisms produce impulse, sound, and tremor on the affected side only, and by this circumstance they may easily be discriminated from aortic enlargements.

In sacculated aneurism seated in the upper parts of the chest, pulsation exists both above and below the clavicles, but I have generally found it stronger below. When the tumor is large, and occupies the left extremity of the arch, the impulse is often perceptible from the sternum to the left shoulder, and as low down as the third or fourth rib. When it lies in contact with the ribs posteriorly, the shock is sometimes felt on the back. This, however, is a rare occurrence.

Dulness on percussion is always found over aneurisms considerably larger than an egg, lying in apposition with the surface.

I searched during ten years for an aneurism immediately behind the heart, with the view of ascertaining whether the presence of this organ in front would or would not disguise the aneurismal impulse and murmur, and render the physical diagnosis impossible. A case at length occurred in St. George's Hospital, which led me to the discovery of a new presumptive sign. The post-mortem appearances were as follows:—The descending aorta, from an inch below the left subclavian down to the diaphragm, was enlarged into an aneurismal sac, which lay across the spine, and projected, on the right side, about three inches beyond the vertebræ, without reaching the ribs; while, on the left, it extended to the ribs, had caused destruction of three and caries of two more, and at last formed a considerable tumor on the back. *The pericardium was adherent to the sac.* Several of the dorsal vertebræ were extensively absorbed. A tract down the front of the sac was formed by the remains of the aorta, a good deal loaded with steatomatous deposition.

" *The heart was slightly enlarged, the left side being a little thickened, while the cavities of the right side were enlarged, without any increase of muscular substance*" (see the Autopsy, drawn up by my colleague, Mr. C. Hawkins, in a work on Dropsy, by Dr. Seymour, p. 15).

The physical signs of this case may be analysed under the several heads of, 1. murmur; 2. dulness on percussion with deficient respiratory murmur, and 3. impulse.

1. The patient was under observation in the hospital for a year. I examined him, for the first time, six months before death. No aneurismal murmur was ever heard by myself or others, either in the præcordial region or on the back: this sign therefore failed. It does not follow, however, that it would fail in every case; for its absence in the present instance might have been referable, not to the interposition of the heart, rendering it inaudible, but to the size, thickness, and configuration of the sac, preventing, as they sometimes will do, the *generation* of the murmur,—a view which is the more probable, because no murmur was audible on the tumor even after it had protruded posteriorly through the ribs. In other cases, similar circumstances preventing the generation of a murmur might not exist: we should, therefore, in all cases, explore for this important sign.

2. On my examination alluded to, six months before death, when there was yet no external tumor, I found dulness on percussion, and deficient respiratory murmur, along the left side of the spine; and Dr. Kingston (who was the first to whom, long before my examination, the idea of aneurism occurred,) had, I believe, observed the same, several months before. Now, as the patient had neither previously had pleurisy, nor then presented the usual physical signs of fluid in the chest; and as he had never had peripneumony to occasion hepatization of the lung, nor then presented any symptom of tubercular consolidation, it followed that the

dulness on percussion and deficient respiration were referable to a tumor of some kind; and considerable pain in the præcordial region, and through to the spine, rendered it *probable* that the tumor was an aneurism,—but not more than probable; since encephaloid tumors in the lungs may create the whole of the same signs; of which I have seen several instances.

3. The impulse of the heart was exceedingly vigorous, and was double, consisting of a diastolic, as well as a systolic impulse, each of a *jogging* character; so that the whole impulse might be called a *double jog*. All the auscultators who saw this case, were agreed that there must be considerable hypertrophy of the heart to account for so strong an impulse: a different opinion, indeed, would have been irrational and unwarrantable, as being opposed to all anterior experience respecting the physical signs of hypertrophy. Yet, as above reported by Mr. Hawkins, the organ was found only "slightly enlarged and thickened."

Now, this strong and double-jogging impulse, in the absence of *adhesion of the pericardium*, and of displacement of the heart to the front of the spine, constitutes the new sign of aneurism, or at least of a tumor, behind the heart, to which I allude. I say, "in the absence of adhesion of the pericardium," because, in the first edition of this work, I pointed out the same double-jog as a new sign of adhesion (see back, p. 194); and I say, "in the absence of displacement of the heart to the front of the spine," because this also occasions the double jogging impulse (see Displacements). The three classes of cases corroborate and throw light upon each other, because they all admit of the same explanation: for as, in adhesion of the pericardium binding the heart down to the spine, and in displacement of the organ to the front of the spine, the ventricles are tilted forward as often as the rounded swell of their body, during the systole and diastole, encounters the resistance of the spine; so the same occurs when an aneurismal or any other tumor behind the heart is the cause of the resistance. It may, therefore, be stated, in conclusion, that in the absence of adhesion, and of displacement, a strong, double-jogging impulse affords presumptive evidence of a tumor behind the heart, the aneurismal nature of which must be determined by the concurrence of other signs of that affection. If there be a murmur in the præcordial region *distinctly not refer-*

able to a valve;—still more, if there be no murmur whatever in the præcordial region, yet one audible on the back, the evidence of aneurism afforded by this sign, in connexion with the double jog of the heart and the posterior dulness on percussion, is almost positive. In the absence of murmur, the signs would not warrant more than presumptious of aneurism, as an encephaloid or other tumor behind the heart might occasion the signs.*

While this is passing through the press, I find that Dr. Todd mentions having "himself observed, some years ago, a case where the heart was pushed forward and outwards, and, as it were, compressed against the ribs, by an enormous aneurism of the thoracic aorta." He makes no remark on the signs, except that "the sounds of the heart were so modified by this compression as to lead to the erroneous diagnosis of concentric hypertrophy." I can easily believe that, if the compression be very great, the sounds may be diminished; for, in my first experiments on the denuded heart of the ass, I found that heavy pressure with the stethoscope on the ventricles, invariably diminished the sounds—of course, by curbing the contractions of the organ and the extension of the valves. It remains, therefore, to be ascertained by further cases whether diminution of the sounds will prove to be a constant sign of an aneurism behind the heart occasioning great pressure.

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* It was supposed by some non-auscultators, that the strong action of the heart in the above case was occasioned by the mere obstacle presented to the circulation by an aneurism at a distance: also, that the absence of dropsy was a proof of aneurism, and of the heart being sound; because, said they, the violent action, if from enlargement, would have been attended with dropsy, whereas aneurism presents so little obstacle to the circulation as not to create dropsy. These statements are contradictory; for if an aneurism presented so little obstacle to the circulation as not to create dropsy, it would not, for the same reason, excite strong impulse of the heart: or, reversing the proposition, if it could occasion strong impulse of the heart, it would, for the same reason, excite dropsy. The facts, however, are incorrect: auscultators know that a remote aneurism, though it may occasion palpitation on exertion, does not morbidly increase the impulse of the heart except by slowly giving rise to hypertrophy: also, that hypertrophy, with violent impulse, may in some cases exist for years without occasioning dropsy, and that, therefore, nothing more than remote presumptions can be drawn from the presence or absence of dropsy in such a case as the present.

SECTION IV.

SYNOPSIS OF THE PHYSICAL, IN CONJUNCTION WITH THE GENERAL SIGNS, IN REFERENCE TO THE SEVERAL VARIETIES OF ANEURISM OF THE AORTA.

Simple Dilatation of the Arch, and ascending Aorta.

Physical Signs.—1. A constant pulsation above both clavicles at their sternal ends; stronger on the right side if the enlargement is confined to the ascending portion, and never communicated to the sternum or ribs, unless the dilatation be enormous.

2. A hoarse rasping murmur, synchronous with the pulse, above both clavicles, of brief duration, commencing and terminating abruptly. If the enlargement is confined to the ascending portion, the sound is louder above the right than above the left clavicle; and, along the tract of the aorta up the sternum, it is superficial, and often of a hissing or whizzing character; by which, and by the murmur being loudest high up the chest, it is distinguishable from that of diseased aortic valves. It is usually distinct on the back, where the venticular sounds, if audible at all, are very obscure.

3. A purring tremor above the clavicles, but never below. It is stronger, and the concomitant sound more grating, in proportion as the interior of the aorta is more overspread with hard, and especially osseous inequalities.

General Signs of Dilatation.—Frequently none. When any exist, they are a slight degree of those common to all organic diseases of the heart, viz. the signs of an embarrassed circulation. They may assume a most aggravated aspect when dilatation becomes complicated with organic disease of the heart.

Fallacies, and Methods of detecting them.

a. *Anæmia* from any cause, (especially in nervous, delicate females,) during arterial excitement, sometimes occasions an impulse and bellows-sound above the clavicles; but they may be discriminated by the impulse being feebler, and the sound more a brief whiff, than in aneurism of the aorta, and by the absence or comparative feebleness of purring tremor. It is, in fact, in the subclavian and carotid arteries that the phenomena take place; for, though the aorta be under the same circumstances of excitement, its action is not so violent as to extend in any appreciable degree to the supra-clavicular regions.

b. *Aortic regurgitation*, particularly when accompanied with much hypertrophy of the heart, I have in many instances found to occasion the impulse and whiffing sound above the clavicles in a still more remarkable degree than anæmic palpitation. The phenomena depend upon the unfilled state of the arteries and the suddenness and energy of the ventricular contraction—a subject already considered (p. 101). They may be distinguished by the sound being more whiffing and less hoarse, and the arterial impulse more *jerking*, than in dilatation of the aorta; but the best criterion is, to ascertain the existence of aortic regurgitation, which may always be done with certainty by the rules given at p. 334.

c. *Dilatation of the pulmonary artery* is a third, though extremely infrequent source of fallacy; for the mode of detecting it I refer the reader to the next head: viz.

Dilatation of the Pulmonary Artery.

Physical Signs.—I have met with one case (Weatherly) in which this artery was dilated to the extent of five inches in its internal circumference. It presented the following physical signs, which have not hitherto, I believe, been noticed. The case of L. P. presents the same, but the patient is living, and the anatomical proof is therefore deficient.

1. A pulsation with purring tremor between the cartilages of the second and third ribs on the left side, and thence in a decreasing degree downwards, *but not appreciable above the clavicles.* Also a slight prominence between the same ribs.

2. An extremely loud, superficial, harsh, sawing sound, audible above the clavicles and over the whole præcordial region, but

loudest on the prominence between the second and third ribs.

The general Signs were those of hypertrophy and dilatation of the heart, which accompanied the dilatation of the pulmonary artery.

Diagnosis from Dilatation and Aneurism of the Aorta.—Dilatation and aneurism of the ascending aorta are perhaps the only affections for which dilatation of the pulmonary artery could be mistaken. The signs, however, of the latter are so characteristic that, with due attention, I think it scarcely possible to commit an error. Namely, a pulsation between the cartilages of the second and third ribs could not possibly be occasioned by a mere dilatation of the ascending aorta; as this artery, even when dilated, is situated too far to the right to extend beyond the margin of the sternum. Again, a sacculated aneurism of the ascending aorta could not reach the cartilages of the second and third left ribs without being large; and in this case it would present dulness on percussion, and form a much greater tumor externally than existed in the present instance. The sound also of such an aneurism would be on a low key, and as if remote, instead of loud and superficial. Finally, a dilatation or an aneurism of the ascending aorta or arch would occasion a pulsation, murmur or tremor above the *right* clavicle or on the right side of the sternum, or above both clavicles,—which was not the case in the instance of which we speak.*

Sacculated Aneurism of the thoracic Aorta.

Physical Signs.—1. A pulsation both above and below the clavicles, but usually stronger below. If the tumor occupies the ascending aorta, its impulse is most perceptible on, and to the right of the sternum. If it is seated in the commencement and middle of the arch, it produces an impulse above and below the right clavicle and about the top of the sternum, often with a visible intumescence of the parts. If it is seated in the commence-pulse and bellows-sound above the clavicles; but they may be discriminated by the impulse being feebler, and the sound more a brief whiff, than in aneurism of the aorta, and by the absence or comparative feebleness of purring tremor. It is, in fact, in the subclavian and carotid arteries that the phenomena take place; for, though the aorta be under the same circumstances of excitement, its action is not so violent as to extend in any appreciable degree to the supra-clavicular regions.

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* Dr. Stokes describes an aneurism "about the size of a goose's egg, which caused a flattish tumor extending from the second to below the third rib, and yielded a pulsation between the second and third left ribs." Here the similitude to dilatation of the pulmonary artery stopped; for there was not any *bruit de soufflet* or *de rape* (Dub. Jour. v. p. 419).

b. *Aortic regurgitation*, particularly when accompanied with much hypertrophy of the heart, I have in many instances found to occasion the impulse and whiffing sound above the clavicles in a still more remarkable degree than anæmic palpitation. The phenomena depend upon the unfilled state of the arteries and the suddenness and energy of the ventricular contraction—a subject already considered (p. 101). They may be distinguished by the sound being more whiffing and less hoarse, and the arterial impulse more *jerking*, than in dilatation of the aorta; but the best criterion is, to ascertain the existence of aortic regurgitation, which may always be done with certainty by the rules given at p. 334.

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Sacculated Aneurism of the thoracic Aorta.

Physical Signs.—1. A pulsation both above and below the clavicles, but usually stronger below. If the tumor occupies the ascending aorta, its impulse is most perceptible on, and to the right of the sternum. If it is seated in the commencement and middle of the arch, it produces an impulse above and below the right clavicle and about the top of the sternum, often with a visible intumescence of the parts. If it is seated in the commencement of the descent, the pulsation and swelling incline to the left side, and sometimes reach even to the shoulder. If it occupy the descending aorta, it is so deeply buried behind the lungs that impulse is never, to my knowledge, perceptible in front; but when the tumor becomes very large and extends backwards to the ribs, it may occasion dulness on percussion and deficient respiratory murmur,—most frequently along the left side of the spine, (see case at p. 447,) and, when erosion of the ribs has taken place, an external pulsating tumor may become perceptible. This, however, is very rare. In front, the pulsation of an aneurism is always stronger on the tumor, than at some point intermediate between it and the heart, and generally stronger than the impulse of the heart itself.

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* Dr. Stokes describes an aneurism "about the size of a goose's egg, which caused a flattish tumor extending from the second to below the third rib, and yielded a pulsation between the second and third left ribs." Here the similitude to dilatation of the pulmonary artery stopped; for there was not any *bruit de soufflet* or *de rape* (Dub. Jour. v. p. 419).

2. The abrupt murmur described under dilatation, but weaker and softer, or less rasping. In large, old aneurisms it has a dull and remote character, and is sometimes louder on the side of the neck opposite to that where the tumor is situated. It is generally audible on the back; and, when the tumor occupies the descending aorta, it is often louder behind, especially on the left side of the spine, than in front. Yet it is sometimes totally absent behind. If, on the back, it has more of the abrupt, rasping sound than the ventricular systole in the præcordial region, the evidence of aneurism is almost positive.

This diagnosis of aneurismal from valvular murmurs is given at p. 442.

3. A purring tremor above the clavicles. I have never found it below, unless the tumor had penetrated through the ribs or sternum; yet I can suppose that a tremor *might* be perceptible through the intercostal spaces, without erosion of the bones. It is weaker than in dilatation, and in old and large aneurisms often becomes extinct. It may be perceptible on the back, near the spine, when an aneurism of the descending aorta has reached the ribs and occasioned an external tumor.

General Signs of Sacculated Aneurism.—Any or all of the following signs may be present. A pulsating tumor, presenting externally, and sooner or later causing livid redness of the integuments; deficient resonance on percussion, and defective respiratory murmur of the part; a sense of retraction of the trachea, with a wheezing respiration and a croaking or whispering voice;

dysphagia; an intense gnawing or terebrating pain in the spine; aching of the left shoulder, scapula, neck, axilla, and arm, with numbness, formication, and impaired motive power of the limb; a sense of weight and infarction in the chest; venous intumescence round the root of the neck, and enlargement of the sternal veins; difference of the two pulses: a strong, double jogging impulse of the heart when the tumor is immediately behind it (see p. 449); some of the ordinary symptoms of organic disease of the heart in a slight degree, but very seldom dropsy.

Fallacies, and the Methods of detecting them.—Pulsation beneath the sternum and ribs, occasioned by amplified glands or other tumors in the anterior mediastinum, by hydropericardium, by enlarged heart, or, finally, by adhesion of the pericardium, may, according to my experience, be easily discriminated from aneurismal pulsation by the following criteria.

a. *Pulsating glands* or other tumors in the anterior mediastinum are not attended with the aneurismal sound, or only in a slight degree; no impulse and tremor are felt above the clavicles; and symptoms of a disturbed circulation either do not exist at all, or do not correspond in severity with the magnitude of the apparent disease.

b. *Hydropericardium*, instead of producing the gradual, steady, and powerful heaving of an aneurism, occasions an undulating motion, of which some of the shocks are stronger than others, and none are exactly synchronous with the sound of the ventricular systole. The undulatory impulse is strongest in the left præcordial region; whereas the impulse of an aneurism of the ascending aorta or arch is situated either on the right side of the sternum or near the top of the sternum and the clavicles on either side, and is notably stronger on the tumor and on the hear, than in the intermediate space. Hydropericardium is not productive of the aneurismal sound. Its history is different from that of aneurism, the latter being very often referred to some injury, or excessive exertion, suddenly followed by faintness, pain, and dyspnœa.

c. *An enlarged heart* produces an impulse which is strongest at the apex, and decreases progressively on receding from it: the beating of an aneurism is stronger on the tumor than at some point intermediate between it and the apex of the heart; and in most instances it is stronger even than the beating of the heart itself. Hence, an aneurism distinctly conveys the impression of there being *two centres of motion*—the tumor and the heart; while the pulsation of an enlarged heart is felt to be referable to one alone. Finally, the ventricular contraction of an enlarged heart produces an ordinary sound, but is not attended with aneurismal murmur, or pulsation above the clavicles. I have never known adhesion of the pericardium to occasion a pulsation which could be mistaken for aneurism, until it had occasioned enlargement of the heart, its ordinary consequence. In this case the diagnostic symptoms are the same as those of enlargement of the heart, with one difference, that the motion is of a more unsteady, double-jogging, and struggling character.

d. *Varix of the jugular veins* above the clavicles, is distinguished by the absence of sound and impulse, and by the compressibility of the tumor. It must not be forgotten, however, that intumescence of these veins, sometimes with immense, springy swelling round the root of the neck, is a sign of a tumor compressing the descending vena cava, and that the tumor may be an aneurism. I have repeatedly seen the swelling result from this cause, and five times from encephaloid tumors of the right lung.

e. *Enlarged glands*, or other tumors, above the clavicles, receiving pulsation from a subjacent artery, rarely occasion sound; and if any exist, it is a feeble whizzing, such as is produced by compressing an artery with the edge of the stethoscope. Both it and the pulsation are confined to the side affected. If the tumor can be grasped, it will be felt not to dilate laterally during the ventricular contraction, and if it can be raised from the subjacent artery, its beating and the whiff will cease entirely.

f. *Subclavian and carotid aneurisms* occasion pulsation, sound, and purring tremor on the affected side alone, and these signs are more superficial and distinct than in aneurism of the aorta. The sound, from not being reverberated through the chest, resembles that of a small hand bellows, instead of having the hoarseness of a forge bellows.*

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* Dr. Stokes describes a very interesting case of an aneurism of the innominata equalling a "large cocoa nut, and in a great part filled with large fibrous and laminated coagula" (Dub. Jour. v. p. 413). He states that it yielded no *bruit de soufflet* or *rape*. This assimilates with the principles above developed (p. 445); that large

g. *Purring tremor* of the chest, proceeding from mucous rattle, may be recognised by its ceasing when respiration is suspended.

Sacculated Aneurism of the Abdominal Aorta.

Physical Signs.—1. A constant, swelling pulsation of extraordinary power.* It appears much stronger to the ear resting on the stethoscope than to the hand. The instrument may be forced down in various directions into close proximity with the tumor, and an idea of its position and dimensions may be thus obtained. By the stethoscope and hand together, it may be readily ascertained that the lateral dimensions of the tumor are distinctly greater than those of the healthy aorta; also, that the *lateral* is generally equal, or nearly so, to the *forward swell;* further, that the tumor is fixed, though compressible, and is commonly of a more or less rounded form.

There are exceptions, however, which should make us cautious in deciding positively against the existence of aneurism by the absence of any of the preceding signs. In a case already alluded to, by Dr. Beatty, a very large aneurism above the cœliac artery occasioned no perceptible tumor or impulse, in consequence of being braced down and pressed backwards by the crura of the diaphragm. I lately attended a case in which there was little or no lateral swell and impulse in consequence of the tumor

fibrinous coagula prevent the murmur. He adds, that the aneurism yielded a "double sound perfectly analagous to that of the excited heart." If this was not a kind of murmur, it must have been the beat of the heart, transmitted through the aorta and the sternum to the tumor.

The disease caused obliteration, not only of the right carotid and subclavian arteries, but of the jugular veins and venæ innominatæ, and induced hemiplegia. The case is principally interesting, however, as having produced several symptoms usually confined to enlargements of the aorta; namely, dysphagia, stridulous respiration, very feeble respiratory murmur of the right lung, and puerile of the left, from compression of the right bronchus; also, dulness on percussion, at first confined to the sternal end of the right clavicle, but eventually pervading "the antero-superior fourth of the right side, the upper third of the sternum, and the sternal fourth of the *left* clavicle."

The descent of so large a tumor into the chest easily accounts for all the symptoms. The diagnosis would be formed with least difficulty in the early stage; namely, by noticing where the disease commenced. Without this knowledge, it would be almost impossible, in the advanced stage, to distinguish such a tumor from an aneurism of the aorta.

* Dr. Stokes has correctly explained the prodigious pulsations of large aneurisms over their whole surface on the principle of the hydrostatic balance.

being braced down by old and firm adhesions of the pancreas across it, and of the kidney on its right side. In St. George's Hospital, about four years ago, was a case of a perfectly *moveable*, pulsating, and compressible tumor, which proved to be an aneurism of the cœliac artery.

2. Dulness on percussion will be perceived if the tumor be large and superficial; but if it be of moderate size or small, (*e. g.* less than a cricket-ball,) I have found that the dulness is neutralised, or, at least, rendered obscure, by the resonance of surrounding or superincumbent intestine, especially if charged with flatus. A purgative, by removing flatulence, will often render the dulness more distinct.

3. A brief and abrupt bellows sound, not so loud or hoarse as that of aneurisms in the chest. It is sometimes audible on the back, but not so often as in pectoral aneurisms. I have, in several cases, heard the murmur loudest at that part of the tumor which I found after death to correspond with the opening into the artery. The murmur is very much limited to the seat of the aneurism, and, from being propagated downwards with the stream in the aorta, it is more audible below than above the tumor.

The murmur is occasionally absent. I found this to be the case in the instance above alluded to, of an aneurism, about as large as a turkey's egg, bound down by firm adhesions of the pancreas and right kidney. I presume that the adhesions prevented the murmur partly by restricting the influx of blood, and partly by thickening the walls of the sac and rendering them unsusceptible of vibration.

Dr. Corrigan has devised an ingenious expedient by which a murmur may often be developed in an aneurism, which did not previously exist: namely, by placing the patient in the *horizontal* position, or even with the abdomen raised higher than the chest. This position, by removing hydrostatic pressure, diminishes the distention of the sac, and consequently permits a freer flow of blood into and out of it; and it is by the inward current that the murmur is occasioned. This device is especially useful in incipient, small aneurisms, before a pulsating tumor is distinctly perceptible. I imagine that great inexpansibility of the sac may prevent its success; for, in my case above described, the murmur did not exist, though the patient was placed horizontally, and was also anæmic, with a pulse of 102. Dr. Corrigan's sign is not to be depended upon alone; because most anæmic subjects yield a murmur when in the horizontal position (see p. 134).

A murmur created by the pressure of the stethoscope on a superficial artery over the tumor, must be carefully distinguished from a murmur of the aneurism itself. This source of fallacy existed in my case above referred to, and I decided that it was superficial by the *nearness* and hissing tone of the sound, by its being restricted to one spot, and by its ceasing whenever the artery was obliterated by firm depression of the stethoscope. It was found after death to have proceeded from the superior mesenteric artery, which descended over the summit of the tumor, and was as large as a quill.

The second sound of the heart is generally inaudible on the abdomen, and, consequently, the pulsation is *simple*. This, however, is a point of little importance.

The physical signs now described, present so many exceptions, and so many other sources of fallacy remain to be noticed, that we must always be slow and circumspect in deciding on the existence of abdominal aneurisms. There are some cases in which it is impossible, without violating the principles of sound inductive reasoning, to give a positive diagnosis. We must then wait and watch.

General Signs.—They are those of slightly impeded respiration dependent on an imperfect descent of the diaphragm; of dyspepsia; of lumbar abscess, with or without caries of the vertebræ and symptoms of spinal pressure; of renal disease; and of pressure on the nerves or viscera of the abdomen and pelvis; but none are pathognomonic of aneurism, except a pulsating and usually compressible tumor, felt through the abdominal parietes.

Fallacies and methods of detecting them.

a *A scirrhous or encephaloid tumor* of the stomach, internally or externally.

b. *Enlargement of the liver* extending across the epigastrium, —which is very common.

c. *Enlargement of the pancreas* by hydatids, or scirrhus—extremely rare affections.

d. *Fungoid or other tumors* of the mesentery, omentum, transverse arch of the colon, or diaphragm, which are pretty common.

e. *Indurated fæces*, air, intestinal concretions, or masses of tape-worm, impacted in the transverse colon.

It is the common property of all these tumors, when they rest upon the aorta, to receive its pulsation, and in many instances to occasion a bellows-murmur by compressing the vessel,—especially when the tumor is pressed down upon it by the stethoscope. Not many years ago, almost every pulsating tumor in the epigastric and umbilical regions was assumed at once to be an aneurism; but modern experience has shown that the great majority are nothing more than the tumors above enumerated. They may generally be discriminated from aneurisms by the following circumstances:—

1. The impulse, with few exceptions, is comparatively feeble; for the elevation of a tumor by the aortic impulse, is not equal in degree or force to the expansion of an aneurismal sac. Diffuse, superficial tumors, as the left lobe of the enlarged liver, I have found to transmit the impulse more feebly than smaller tumors sitting immediately on the aorta, as an enlarged pancreas.

2. The impulse is still more feeble, and sometimes imperceptible, when the stethoscope is applied *laterally;* since aneurisms alone present a considerable lateral expansion. When the tumor can be displaced by lateral pressure, so as totally to lose its impulse, (especially if the aorta can then be felt to be of its natural size,) the evidence against aneurism is almost positive. If, after the displacement, the impulse continue undiminished, an aneurism of the cœliac, its branches, or the superior mesenteric artery, may be suspected. The enlarged liver yields no lateral impulse. The lateral impulse is best examined by inclining the patient a little to the opposite side, while he lies in the horizontal position.*

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* Professor Harrison of Dublin states that he has met with, or seen, aneurisms of the cœliac, hepatic, splenic, gastric, and mesenteric arteries, of the left gastro-epiploic, the coronary of the stomach, the right spermatic, and the left renal capsular (Dub. Jour. v. 436).

Dr. Stokes met with an aneurism of the hepatic artery, the size of a large orange, and pressing directly on the bile duct. It was covered by the capsule of Glisson and by the pancreas, which encircled its lower half. He did not detect any pulsation in

3. The tumor, when connected with the stomach, colon, or omentum, is often superficial, and moveable with the movements of these viscera, so as to lose all impulse, both lateral and direct.

4. A few brisk purgatives of calomel, colocynth, and aloes, will often remove indurated fæces, intestinal concretions, and flatulence, and thus dissipate the tumor and impulse. The same remedies, followed by ounce doses of ol. Terebinth. purif., will often produce a similar effect on masses of tape-worm.

5. Tumors, if solid and firm, are more incompressible than the generality of aneurisms: yet this sign is of little value, because many tumors, as the encephaloid and flatulent varieties, are very springy and compressible; while, on the other hand, I have several times found aneurisms wholly incompressible, in consequence either of the thickness of their sacs, or of the firm adhesion of the pancreas, kidneys, crura of the diaphragm, or other contiguous parts.

6. In cases of enlargement of the liver, dulness on percussion extends, *without any interval*, from the right hypochondriac region and scrobiculus cordis, over and beyond the seat of the pulsation; and the outline of the viscus may finally be traced with the fingers. Except in reference to the liver, dulness on percussion must not be too much trusted; as it may result from other solid tumors besides aneurisms, and it may be absent or indistinct in aneurisms of small, and even moderate dimensions, in consequence of the resonance of superincumbent or surrounding intestine.

7. The murmur of an ordinary tumor is generally less than that of an aneurism, being only a slight whiff, like that produced by compressing an external artery; and it may often be suspended by applying the stethoscope laterally and pushing the tumor off the aorta,—which is not the case with an aneurismal murmur. It must not be forgotten that an artery running *over* any tumor may create a superficial murmur when the vessel is

it, and he ascribes the absence of the phenomenon to the want of counterpressure beneath. The tumor and the enormously distended gall-bladder had, within a brief period, protruded the liver downwards, imparting to it the aspect of great enlargement (Dub. Jour. v. p. 402). The same protrusion occurred in Dr. Beatty's case of aneurism (Dub. Hosp. Rep. v.) These facts are worth recollecting in reference to the diagnosis of obscure diseases of the liver.

compressed with the stethoscope. Such a murmur, therefore, must not hastily be considered aneurismal.

8. Collateral evidence for, or against aneurism, is sometimes afforded by the history and general symptoms. Thus, malignant disease in other parts, with general cancerous cachexia, would afford presumptions that an abdominal pulsating tumor was malignant. Existing and previous hepatic derangement would countenance the view of enlargement of the liver. Though derangement of the stomach and bowels, with constipation, flatulence, gnawing pain, &c. would direct the attention to those organs; yet such signs are treacherous, because it has been shown that an aneurism, by irritating the cœliac plexus and other abdominal nerves, may occasion the utmost degree of functional disturbance. If the patient be young, as, for instance, under æt. 20 or 30, of healthy constitution and family, and have never experienced sudden and great faintness and dyspnœa, with or without pain, after any considerable corporeal exertion, as running, ascending a hill, lifting, straining, rowing, gymnastics, &c., the presumptions are against aneurism; and *vice versâ*.

Cases must be considered doubtful when the signs are partly those of aneurism, and partly of an ordinary tumor. The following case will exemplify how many important signs of aneurism may be absent, yet the disease exist. A gentleman in Scotland, whom I saw in consultation with Dr. Abercrombie, and Drs. Munro and Chisholm of Inverness, presented a rather strongly pulsating tumor in the epigastric region. He was under æt. 28, and of remarkably healthy constitution and family, and could not recollect to have felt suddenly ill after corporeal exertion. There was scarcely any lateral expansion and impulse; no murmur, even in the horizontal position, except the whiff of a superficial artery, created at pleasure, in one spot, by pressure with the stethoscope; no distinct dulness on percussion; no pain in the epigastric region or back, except a little occasionally, and at long intervals of weeks or months; no disturbance of the circulation,—for, a month before I saw him, he had ascended a high mountain in an unusually short time; p. 100, with emaciation and anæmia, since cerebral convulsions and active treatment a fortnight before my visit; no signs, physical or general, of disease of the heart or lungs, and they were ultimately found healthy. There were severe dyspeptic symptoms, with constant craving, constipation, often bilious evacuations, and emaciation,—symptoms which had existed for about a year and a half, and which seemed to point rather to a tumor connected with the stomach, bowels, or pancreas, than to an aneurism. Opposed to these symptoms, so little indicative of aneurism, stood the single, but important sign of a rather strong, direct pulsation of the tumor,—a pulsation, however, not stronger than I have seen from ordinary tumors. Under these circumstances, it was agreed that the symptoms did not justify a positive opinion, that the case should be considered doubtful, that it would be necessary to wait and watch, and that, meanwhile, the treatment should be conducted on principles embracing both views.

The patient died a fortnight afterwards, from rupture of the aneurism, and extravasation of five pints of blood into the cavity of the abdomen. The post-mortem appearances, to which I have several times alluded, fully explained the symptoms, or, rather, the want of them. The sac (exclusive of external coagula formed after its rupture) was three inches long by two broad; and it sprang from the right side of the aorta, by an aperture as large as a shilling, half an inch below the cœliac artery. The deficiency of lateral pulsation was attributable, not only to the moderate dimensions of the sac, but also to extensive, old adhesions of the pancreas on the left and front of the tumor, and of the kidney on its right, which firmly bound it down. The absence of murmur was referable to the limited expansibility of the tumor, resulting from the same causes; and these also accounted for the patient's capability of great exertion, without *apparent* inconvenience, so late as six weeks before his death. The superficial whiff proceeded from the superior mesenteric artery, which crossed the summit of the tumor. The absence of dulness was referable to the stomach, constantly distended with flatus, being adherent by its pyloric extremity to the most prominent part of the tumor. The absence of all but slight and occasional pain was accounted for by the uninjured state of the spine. The dyspepsia was connected with mamellated thickening of the mucous membrane of the stomach from chronic inflammation, probably excited by the irritation of the tumor.

The absence of so many important symptoms of aneurism, strongly evinces the necessity for caution in all such cases. The only safe course is, to adhere rigidly to the rules of inductive reasoning, and never to draw *positive* conclusions from evidence which is merely *presumptive* or *equivocal.* If the practitioner allow vague impressions and undefined convictions to divert him from this course, he cannot fail to commit occasional errors in diagnosis, of which the patient, no less than his own reputation, may have to pay the penalty; for the latitude in diet, exercise, &c. which might be admissible in the case of ordinary tumors, would be destructive in aneurism; while the restrictions unavoidable in the latter, would often be detrimental to the general health in the former. By treating an equivocal case as doubtful, till it has declared itself, both extremes may be avoided.

Anæmic and nervous Pulsation of the Abdominal Aorta.—The illustrious father of auscultation ascribed this phenomenon to nervous and hysterical irritability, with spasm of the aorta; but I have shown that anæmia—a deficient, or too watery state of the blood, is its most essential constitutional cause, while nervousness, whether pre-existent or consequent, co-operates by accelerating the circulation (See Inorganic Murmurs).

This is a very frequent and deceptive affection, leading the unwary to the supposition of aneurism. When, says Laennec, it exists in conjunction with air, pent up in the colon or duodenum, and presenting the feel of a compressible tumor, the resemblance to aneurism is still more complete. The aortic throb will also increase the pulsation of any solid tumors resting on the aorta, as described under the preceding head. After an examination of many cases, I am satisfied that, in the absence of immovable, solid tumors resting on the vessel, attention to the following circumstances will render the diagnosis easy.

Physical Signs.—The cylinder may be pressed down on the aorta, so as to yield a distinct feel of the vessel of its natural calibre. The sphere of its pulsation is limited transversely, but extensive longitudinally, being usually more or less perceptible from the epigastrium to the bifurcation. The impulse, instead of being the gradual, steady, and irresistible heaving or expansion of an aneurism, is a smart, though vigorous jerk; and the sound, when any exists, is merely a short whiff, distinguishable by its shortness from venous murmur, and audible along the whole course of the vessel, instead of being loudest at one spot, as in aneurism. Dr. Graves has shown that it may sometimes be excited by the horizontal position, when, from hydrostatic pressure, it does not exist in the erect. Inorganic murmurs and thrill, with a jerking pulse, in the carotid and subclavian arteries, and venous murmurs in the jugular veins, generally coexist with aortic pulsation, and serve to corroborate the diagnosis.

The *general symptoms* are anæmic, nervous, or hysterical; and the pulsation and murmur are of an inconstant character, increasing and diminishing with the exacerbations and remissions of the arterial excitement.

Aortic Pulsation from Enteric Inflammation.—This has been pointed out by Dr. Stokes. "There is," says he, "a pulsation of the abdominal aorta or its immediate vessels, which is symptomatic of inflammatory disease in the digestive system, and which a long experience enables me to say may be considered an important assistance in diagnosis. A throbbing, generally commensurate with the disease; removed by treatment calculated to relieve enteric inflammation, and aggravated by everything which will increase this affection. In other words, we may have, from enteritis or peritonitis, a throbbing of the abdominal aorta or its vessels, perfectly analogous to the morbid action of the radial artery in whitlow, or of the carotids or temporal arteries in cerebritis." The cases in which he has most frequently observed this symptom, are those of the gastro-enteric fever of Ireland; also, in cases of fever after corrosive poisoning, where the pulse was almost absent at the wrist; and in peritonitis, where no pulse could be felt. In several instances, this want of proportion between the action of the radial, and the abdominal arteries, *combined with fever*, was the principal indication of enteric disease. He has found the increased action extend along the iliac, to the femoral arteries (Dub. Jour. v. p. 438). I have not yet had an opportunity of verifying these observations so extensively as I could have wished; but I think them deserving of much attention, both because they are consistent with analogy, and because they emanate from so accurate an observer as Dr. Stokes. One precaution would be requisite: namely, as many of the febrile cases in question are anæmic, it would be necessary to make sure that the pulsation was not merely anæmic, rather than inflammatory.

APPENDIX TO ANEURISM OF THE AORTA.

I have met with a case in which an aneurismal pouch of the aorta burst into the right ventricle; and Dr. David Monro of Edinburgh has favoured me with the particulars of another case, in which a dilated and diseased aorta burst into the pulmonary. As such cases present peculiar signs, and as their diagnosis has not, to my knowledge, been hitherto explained, I need no apology for introducing them here.

Case of an Aneurismal Pouch of the Aorta bursting into the right Ventricle (Fig. 21.)

John Mitchell, æt. about 25, baker, admitted into the Westminster Hospital, where I saw him, October 27, 1837, by the politeness of Dr. Roe, and Mr. Thurnam, the resident apothecary. He stated that he had felt perfectly well till nine weeks previous to my visit; when, on lifting a sac of flour, he felt a "creak in the heart," and became faint and very pale.* Though "very ill," he continued at work for three or four days; when he gave up and got bled. A fortnight after the accident he entered the hospital.

I made the following notes on the day of my visit. Face slightly bloated, and of purplish or venous tint; legs very œdematous; hands slightly; p. 80, *singularly "jerking,"* especially in the carotids. I think I have never felt a pulse equally jerking. It was like a hard ball, forcibly shot through the vessel. Does not complain of pain. The effort of drawing his flannel waistcoat over his head caused shortness of breath, and intermittence and irregularity of the pulse, for two or three minutes.

Physical Signs.—Dulness on percussion over an extent of about three inches in diameter, extending nearly up to the third rib.† A *marked, superficial* purring tremor over the *upper* portion of the dull part, most perceptible about two inches from the sternum, in the intercostal space. At the same part, there is a *superficial, very loud* sawing sound, like a whispered *r*—more intense during the systole of the ventricles; also a slighter second whizz accompanying the second sound. Moreover, there is *a continuous rumble, through which the other two murmurs are heard.* These sounds are audible, but *less distinctly,* over nearly the whole remainder of the dull part. No purring tremor or murmur above the clavicles; the second sound is there very feeble—indeed barely audible, and the first is wholly inaudible. *Impulse* of the heart not materially increased.

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* See p. 198 for instances of rupture, with similar symptoms: also, the case of Williams delineated in Fig. 13.

† Mr. Thurnam found the dulness and tremor ascend to the second rib. This was probably because he examined him in the horizontal position, whereas my examination was made in the semi-erect position.

The treatment was principally diuretic. The anasarca increased to a great amount, and the patient died about three weeks after my visit.

Diagnosis.—This case was so singular, that I could only give doubtful and conditional diagnosis. I conjectured that a valve or chorda tendinea had been ruptured by the lift, this being denoted by the "creak in the heart," and the sudden paleness and fainting. I felt certain that there was free regurgitation out of the aorta; as it was positively indicated by the peculiarly jerking pulse, by the whizz with the diastole, over the semilunar valves, and by the almost complete extinction of the second sound above the clavicles, denoting that reaction on the aortic valves was defective. Mitral regurgitation also was indicated by the loudness of the first murmur near the apex of the heart. The continuous rumble and the strong tremor, however, remained to be accounted for; and as I had once seen these signs occasioned, in pericarditis, by friction of lymph and churning of a moderate quantity of fluid (case of Jones), I thought the same might be the case in the present instance, assuming endo-pericarditis to have been excited by the rupture. Though the existence of aneurism was possible, there was no direct proof of it; and, if it existed, it was under some new combination of circumstances; as the continuous murmur is foreign to ordinary aneurisms.

Autopsy.—The right cavity of the chest contained several pints of serum, by which the heart was displaced to the left side. Little serum in the left cavity. Two or three ounces of clear serum in the pericardium, and a few scattered patches of recent lymph on its surface, which were easily peeled off with the back of a scalpel (Pericarditis). Heart of natural size and thickness. *Mitral valve* thickened and opake; its chordæ tendineæ thickened and shortened, one being nearly as thick as a crow-quill. *Aortic* *valves* similarly thickened, but in a less degree: both presented recent, florid granulations or vegetations (Endocarditis). The *aorta*, immediately above its valves, was dilated into an aneurismal pouch as large as a small hen's egg, which presented directly forward towards the mouth of the right ventricle, where it formed a tumor, opening, by two apertures on its summit, into the cavity of the ventricle, immediately below its valves (See Fig. 21, *e*). One aperture was as large as an average pea; the other, half the size. The origins of two of the pulmonic valves were separated about a quarter of an inch, in consequence of the interval having been stretched by the subjacent aneurism. The valves, therefore, necessarily admitted of regurgitation.

Remarks.—I should explain the signs of this case as follows: The systole of the left ventricle caused regurgitation through the aneurismal apertures into the right ventricle,—the resistance in this direction being less than that offered by the aortic circulation. The simultaneous systole of the right ventricle expelled a stream, which was not only tilted forwards by the aneurismal tumor, but encountered the stream of regurgitating blood already playing directly forwards against the front of the right ventricle, near its mouth:—which part, being thus thrown into strong vibration, yielded the purring tremor; while the agitation and friction of the blood occasioned the concomitant loud, *superficial*, sawing sound. These phenomena were the most intense about two inches to the left of the sternum, between the third and fourth ribs, because the mouth of the right ventricle was displaced thither by the fluid in the right cavity of the chest. Such being the explanation of the phenomena during the ventricular systole, we next proceed to the diastole. During this, there was a free regurgitation from the aorta, through the aneurismal apertures, into the right ventricle; and also from the pulmonary artery, through the interval between the origins of the two pulmonic valves seated on the aneurism. The two streams thus met and were directed forward against the front of the right ventricle, by exactly the same circumstances as during the systole: hence the murmur and tremor were maintained, though with less intensity, during the diastole also. The *natural* second sound was almost extinct, 1st. because the reaction of the aortic blood on the semilunar valves was enfeebled by the aneurismal regurgitation: 2d. because the regurgitation between the pulmonic valves prevented the due expansion of those valves also.* Such was what occurred during the ventricular diastole. But there was, further, a continuous rumble, occupying all the intervals between the systolic and diastolic murmurs. This appears to me to have been occasioned by the aneurismal regurgitation being incessant; the predominant pressure, first, of the left ventricular contraction, and, next, of the aortic reaction being in incessant operation.

The state of the mitral valve verified the diagnosis of regurgitation through it.

The following case, obligingly given to me by Dr. David Monro of Edinburgh, strongly corroborates the preceding.

Case of Rupture of a Dilated Aorta into the Pulmonary Artery.

James Evans, æt. 24, a porter, admitted into the Edinburgh Infirmary Oct. 30th, 1833. Accustomed, from his profession, to lift heavy weights. Had a severe attack of acute rheumatism about ten years ago. About ten months ago had an attack of pneumonia, which yielded to copious depletion. To this he ascribed his symptoms: viz. palpitation, dyspnœa, followed, three months before admission, by swelling of the abdomen and lower extremities, which has gradually increased.

On admission, the following were the symptoms. Great dyspnœa, amounting to orthopnœa; abdomen much distended, and fluctuation; lower extremities swollen and tense; countenance tumid, and somewhat *livid;* great general uneasiness; action of the heart tumultuous; diffused over a large space, not *strong;* cough with expectoration; *pulse large, harsh, and thrilling*, 112. *Physical signs.*—Much dulness on percussion in the præcordial region. First sound accompanied by a loud soufflet, audible over the whole fore part of chest, and on the back on both sides of spine, but most distinct at the middle of sternum. Second sound short, and much obscured by the first (hence it appears that a *continuous* murmur extended from the first, over the second sound).

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* This is one of the best pathological cases that I have met with, to prove that the semilunar valves are the cause of the second sound. Had one set of the valves only been disabled, the second sound, produced by the other set, would have been distinctly heard near the clavicles, where it is transmitted unobscured by any murmur which might conceal it nearer the valves. But in this case both sets of valves were almost incapacitated: accordingly, the sound was almost extinguished at the clavicles.

The treatment employed, viz. digitalis, calomel, and squills, had the effect of reducing the pulse and increasing the quantity of urine; but produced no impression on the symptoms.

His general uneasiness continued, though temporarily relieved by a small bleeding. The pulse became intermittent some days before death, which happened a fortnight after admission.

Autopsy. Much anasarca. *Chest.* Several pounds of serum in both pleuræ. *Heart,* enveloped in the pericardium, occupied a great part of left side, displacing the corresponding lung. It was found to be more than twice the natural size, pale, flabby, and blunt towards the apex. All the cavities were much dilated, together with the corresponding orifices. The walls of both ventricles retained their natural thickness. All the *Valves* healthy, excepting the semilunar at the mouth of the aorta, which were thickened. The *Aorta* itself, from its origin to the arch, was dilated into a large, irregular sac, which adhered firmly to the pulmonary artery, and communicated with it by two openings, situated an inch and a half from the valves;—the largest, capable of receiving the point of the little finger; the smaller, of transmitting a crow-quill. The edges of both were regular, round, and cartilaginous. Nearer the arch, a third small opening was dicovered, with thin, rugged edges. The internal membrane of the dilated portion of the aorta was reddened and rugous, from numerous cartilaginous patches, which had advanced in some parts to ossification.

Remarks.—The two foregoing cases corresponded in the following particulars. 1. A lift was the immediate cause of the symptoms, though disease of the aorta had preceded.

2. Pulse pre-eminently jerking; for such was evidently the "*large, harsh* and *thrilling*" pulse of Dr. Monro's case.

3. A loud, superficial murmur with both sounds, incessant in one case, and apparently so, judging from Dr. Monro's description, in the other.

4. A livid, venous tint of the complexion.

5. Great, rapid, and universal dropsy.

From these data, I should consider the following to be the pathognomonic signs.

Signs of Aneurism of the origin of the Aorta opening into the Right Ventricle.

Physical Signs.—1. A remarkably loud, harsh, superficial, sawing murmur with both the systole and diastole, together with a continuous, *incessant* rumble; both most audible above the level of the fourth rib, on or near the sternum, and thence, along the tract of the pulmonary artery, up to the interspace between the second and third rib. (Great care should be taken to ascertain whether the heart is displaced to either side by fluid or other causes; as this was the main source of obscurity in the case of Mitchell).

2. A purring tremor in the same situations. It would perhaps not be very perceptible between the third and fourth ribs, if the heart was not displaced, the base of the right ventricle being naturally a good deal covered by the sternum; but, by causing the patient to lie inclined towards his left side, and thus displacing the heart, the tremor would, I have no doubt, become perceptible. It would, of course, be more marked when the lung, a bad conductor of sound and tremor, is displaced from the anterior surface of the heart by hydropericardium (as in Mitchell's case) or by enlargement of the organ.

3. Weakness or extinction of the second sound, near the clavicles, in consequence of the reaction of the aortic blood on the valves being enfeebled by the regurgitation. If both sets of semilunar valves happened to be implicated, (as in Mitchell,) the sound might be almost, or wholly extinguished.

General Signs.—1. Pulse pre-eminently jerking, in consequence of free regurgitation out of the aorta.

2. Great, rapid, and universal dropsy, resulting from general venous retardation, occasioned by the pressure of the aortic circulation being thrown on the right ventricle, and constituting a formidable impediment to the transmission of its blood.

3. A livid, venous complexion, partly from the cause last specified, and partly from a proportion of arterial blood being delivered to the lungs, to the exclusion of an equal quantity of venous:

whence the total amount of arterialized blood in the system is diminished.

4. If the symptoms followed a lift or effort, producing sudden faintness and paleness, the evidence would be stronger.

Signs of Aneurism of the Aorta opening into the Pulmonary Artery.

Physical Signs.—1. A very loud, superficial, sawing murmur, prolonged continuously over the first and second sounds (and probably weaker during the interval of repose): loudest along the tract of the pulmonary artery.

2. A purring tremor in the pulmonary artery, in the interspace between the second and third ribs.

3. The second sound weakened at the clavicles.

General Signs.—1. The jerking pulse.

2. Great, rapid, and universal dropsy.

3. A livid, venous tint.

4. The circumstance of the symptoms having followed an effort, would afford corroborative evidence.

Diagnosis of Aneurism of the Aorta opening into the Right Ventricle or the Pulmonary Artery, from other Diseases.

Dilatation of the pulmonary artery presents a murmur with the first sound only, and the pulse is not jerking. The complexion is not livid, and dropsy may not supervene for years (see case of Weatherly, exemplifying all this).

Contraction of the pulmonic valves with regurgitation (extremely rare). A loud, superficial murmur attends each sound; also purring tremor; but there is no continuous murmur in the intervals, and the pulse is not jerking (case of Rogers).

Contraction of the aortic valves with regurgitation.—A murmur with each sound; but not nearly so loud or superficial, from the aorta being more deeply seated; no continuous murmur; little or no purring tremor; p. jerking; complexion not livid, and dropsy may not supervene for years.

*An aneurismal passage from the origin of the aorta into the left ventricle.**—P. is jerking. A murmur might attend each sound, the first being occasioned by disease of the aorta or its valves, and the second by regurgitation through the passage; but the murmurs could not be incessant, because the regurgitation could only exist during the diastole; nor would they be so loud and superficial as in the above cases.

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* See "Real Aneurism of the Heart," p. 320, for cases of this kind.

Congenital contraction of the pulmonary orifice, and a common opening of the right and left ventricles into the aorta, below its valves. (Case of Mary Collins. See Malformations.)—A very loud, hissing, *superficial* murmur with the first sound only, loudest about the middle of the sternum, over the affected orifices: pulse not jerking, but very small and weak. I have met with two other cases of cyanosis (still living) similar to this, except that in one—Master R.—the second sound was accompanied with a very slight filing murmur from regurgitation.

Friction of lymph with churning of a little fluid in the pericardium; also inflammatory disease of the aortic and mitral valves, with regurgitation through both. (See the remarkable case of —— Jones).—Here, there was a murmur with both sounds, a continuous rumble, a purring tremor and a jerking pulse; but the rumble and tremor (which I ascribe to the rubbing and churning of the lymph and fluid) were equally diffused over the whole front of the ventricles, and were less distinct up the pulmonary artery; no lividity, and little dropsical tendency.

SECTION V.

SPONTANEOUS CURE AND MEDICAL TREATMENT OF ANEURISM OF THE AORTA, AND TREATMENT OF NERVOUS PULSATION.

Previous to entering upon the treatment of aneurism of the aorta, we shall advert to the mechanism by which its spontaneous cure is effected; as the reader will thus be better enabled to understand the principles on which the treatment is founded.

The movement of the blood within the sac being retarded, partly by the roughness of its internal surface, and partly by the fluid being withdrawn from the direct channel of the circulation, coagulation takes place, and fibrine is deposited and organized in successive strata, until the cavity is at length completely filled. The sac, being then no longer exposed to the distensive pressure of the circulation, tends to contract by its own resilience and the compression of the incumbent parts, partial absorption of its contents takes place, and the aneurism is finally reduced to a small, dense, flesh-like tumor. In arteries of the second, and inferior orders, the coagulum generally extends to, and obliterates the calibre of the vessel itself;* but this is rarely the case in the aorta, as the force of the circulation in so great a vessel prevents the lodgment of coagula. Instances, however, of obliteration of the aorta by fibrine when its coats were diseased, are not without example: an important case has been published by Professor Alexander Monro,† and Dr. Goodison describes another.

It is principally in false and mixed aneurisms that the cure by deposition of coagula takes place. In true aneurism, and in dilatation, such a cure is very rare; for, the walls being unbroken and smooth, and the aperture of communication with the sac being in general large, the blood is seldom arrested to such a degree as to deposit lamellated coagula. When, however, the whole circumference of an artery is converted into a bony cylinder, there is a great tendency to its obliteration by a plug of fibrine. Dr. Goodison's case was of this description, and I have more than once seen the same in arteries of the second order.

Hence, as the formation of coagula within the sac is the principal means employed by nature in effecting the cure of aneurisms, the primary object of medical treatment is, to promote the deposition of coagula; and we now proceed to consider the means by which this may be best accomplished.

The antiphlogistic treatment, rigorously pursued, acquired great celebrity as the most efficient remedy for aueurism of the aorta, under the designation of the treatment of Albertini and Valsalva. By detraction of blood and spare diet they reduced their patients to so extreme a state of debility that they were scarcely able to raise their arms from the bed. Morgagni reports[*] that when Valsalva had taken away as much blood as was requisite, he made it a custom to diminish the quantity of meat and drink more and more every day, till he proceeded so far as to allow only half a pound of pudding in the morning, and in the evening half that quantity, and nothing else except water, and this also within a certain weight. After he had sufficiently reduced the patient by this method, so that from weakness he could scarcely raise his hand from the bed, in which he lay by Valsalva's order from the very beginning of the disease, he increased the quantity of aliment by degrees every day until the necessary strength returned. Pelletan, who followed this treatment, sometimes allowed two basins of broth in twenty-four hours, and lemonade as a common drink. Laennec recommends the energetic employment of the treatment of Valsalva (tom. ii. p. 742).

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* Vid. Hodgson, Jones, Farre, Baillie, Petit, Desault, Scarpa.

† Observations on Aneurism of the Abdom. Aorta by Professor Monro, Ed. p. 5 and 8, 1827.

In the first edition of this work I pointed out the numerous difficulties which surrounded this treatment, and showed that it was only applicable to a very limited number of cases. Further observation since that time has served to increase my conviction both of its danger and its inefficiency. In persons of very feeble constitutions, it is utterly inadmissible; since it might be directly fatal by inducing irremediable sinking, or indirectly, by establishing a state of anæmic debility, from which the patient could never afterwards completely rally. When organic disease of the heart complicates the aneurism, the treatment is equally inadmissible; since excessive blood-letting is apt to induce alarmingly protracted, and sometimes immediately fatal syncope. The treatment, again, could not have a curative effect on any aneurisms except those of the false, or the mixed species,—namely, by rupture of the arterial coats; or, if of the true species, such as have a sac so deep, and with so narrow a neck, as to be considerably removed from the direct current of the circulation; for in scarcely any others do laminated coagula form, whatever be the mode of treatment employed.

Even in the few cases which remain after the abstraction of the above, I strongly doubt whether the treatment really promotes the coagulation of blood within the sac. After a certain amount of blood-letting in healthy, vigorous constitutions, *reaction* is induced,—a phenomenon which, by producing an inordinate activity of the circulation, counteracts the desired effect of the depletion, and increases, instead of diminishing, the pulsation of the tumor. Of the reality of this reaction I can entertain no doubt, both from extensive observation on the human subject, and also from the experiments on dogs described at p. 100. In these animals, bleedings, repeated daily or every other day, occasioned, after three or four abstractions, the most violent arterial throbbing. Even in weakly individuals, who have not sufficient constitutional vigour to give rise to much violence of reaction, the circulation is, notwithstanding, accelerated by the anæmic state induced by the bleeding, and the pulsation of the heart and of an aneurism is correspondingly increased. In both classes of patients—the robust and the weakly, the blood, after repeated abstractions, becomes very serous, of a pale crimson, instead of the natural dark venous colour, contains only one-sixth to one-twelfth of the natural proportion of crassamentum, and has sometimes a whitish cream on its surface after standing twelve hours. As such blood contains only a very small proportion of fibrine and red globules, it is ill adapted for the formation of fibrinous coagula; and this circumstance, in connexion with the increased pulsation of the tumor attending the anæmic state, appears to me to afford the strongest reason for believing that the treatment in question is pernicious, rather than salutary. It may be fairly questioned, indeed, whether the treatment of Albertini and Valsalva has ever really merited the reputation which it acquired. For, as the diagnosis of aneurisms of the aorta was involved in deep obscurity until the last ten or fifteen years, it is pretty certain that many cases reported as cured, were really not aneurisms, but other tumors, or anæmic and nervous pulsation of the aorta, simulating that disease. This is probably one strong reason why the treatment in question has been unable to maintain its ground. But there is another; namely, its severity. Though patients will submit to rest and extreme abstinence, they have rarely fortitude enough to permit the superaddition of blood-letting. The practitioner, on the other hand, has seldom the courage to insist upon it, knowing that it is not exempt from danger, and that it will not necessarily be productive of a cure. I must frankly avow that, were I personally the subject of aneurism, I would rather take the chances of the disease, than of the treatment.

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[*] Epist. xvii. art. 30.

In the first edition of this work, I proposed a new treatment for hypertrophy of the heart, opposed to that of Albertini and Valsalva, and their supporters, Laennec, M. M. Bertin and Bouillaud, &c. I had found that excessive blood-letting, by inducing the anæmic state, increased palpitation, favoured the supervention of dropsy, and hurried the case to a fatal termination. On the contrary, I found that moderate bleedings at long intervals, as six or eight ounces every three to six weeks or more, reduced the action of the heart without diminishing the fibrinous quality of the blood. The decided success which has attended this treatment of hypertrophy, has led me to apply it to aneurism of the aorta; and the results have been far more satisfactory than any that I have witnessed from the profuse bleeding system of Albertini and Valsalva. Others seem to have made similar observations. Thus Dr. Beatty remarks, respecting his case reported in the fifth vol. of the Dub. Hosp. Rep., that the patient experienced relief, when he changed from a reducing system to a more nutritious and generous diet. Dr. Stokes has made a similar remark on another case, in the Dub. Jour. vol. v.

In conformity with these principles, the treatment, in my opinion, should be as follows: The patient should, in the first instance, be pretty copiously bled, from twelve to twenty ounces being drawn, according to the age and strength. After this, it will generally be sufficient to abstract ℥vi or viii every three to six or more weeks, the quantity being the larger, and the interval shorter, in those who are robust and plethoric, and who speedily reproduce blood. An increase in the strength of the pulse, and of the pulsations of the tumor, should be the signal for the depletion. But when the first signs of anæmia display themselves by slight paleness of the complexion and lips, a little jerk in the pulse, a sense of palpitation of the heart, and a feeling of general debility, bleeding should be entirely suspended till this state has been completely removed; for it indicates that the depletion has already been carried a little too far.

Purgatives and diuretics may be made to co-operate with venesection. It might be imagined that purgatives alone would suffice to reduce the mass of fluids in the system, without the aid of blood-letting. Experience, however, has convinced me that this cannot always be accomplished without a degree, and a continuance, of purgation highly intolerable to the patient, and not exempt from the danger of permanently injuring the mucous membranes. Occasional purgation, however, continued for a week or ten days at a time, may be resorted to with great advantage after blood-letting; as it keeps down the quantity of the blood, without depriving it to the same degree of its fibrine. In this view, the purgatives which produce aqueous evacuations, are the most suitable. The neutral salts will suffice for ordinary occasions; but when a powerful effect is required, nothing is comparable to elaterium, by which two or three pints of serum, or more, may sometimes be drained away in twenty-four hours. Jalap and bitartrate of potass have in a less degree the same effect. Diuretics may be given on the same principle. These remedies, no less than bleeding, should be employed short of that degree which would produce anæmia.

Digitalis is eminently useful in the treatment of aneurism, by enfeebling and retarding the action of the heart and arteries, and thus promoting the stagnation of blood within the sac. So decidedly has it this effect, that I have found it a dangerous remedy in organic diseases of the heart attended with great debility of the organ; since it is apt to prove fatal by creating polypus (see Polypus). In aneurism, the patient, if suitably watched, may be kept moderately under its influence for several consecutive weeks, when an interval of a week or two may be interposed, to obviate any cumulative poisonous effect. If the heart be simultaneously affected with dilatation and attenuation, softening, or great valvular disease, the omissions should be at shorter intervals, and the doses should always be moderate, for the reason above assigned.

The well-known effect of the acetate of lead in controlling active hæmorrhages, has introduced this as a remedy for aneurism. In Germany it has been extensively used for many years, and Dupuytren, Laennec, and Bertin have employed it with advantage in France. My own experience is in its favour. It may be given occasionally, where digitalis disagrees, or when the patient tires of that remedy, or takes a prejudice against it. Its tendency to produce inflammation of the mucous membrane of the stomach and bowels may be counteracted by conjoining it with opium, or, as Dr. Thompson has pointed out, with vinegar. I have seldom found inconvenience from a grain, with half a grain of opium, in a pill, given three or four times a day. So small a dose, however, is insufficient to produce a full effect. For this reason, and also because opium is a stimulant to capillary action, the formula with vinegar is perhaps preferable. Two or three grains of the acetate, in a pill, may be safely given every four hours, provided it be washed down by a draft containing half an ounce of common vinegar, or an equivalent quantity of strong acetic acid. Sometimes the vinegar itself irritates; but this may be in a great measure obviated by a liberal addition of sugar, or, if this fail, by tr. opii m iv or v with each dose. If, notwithstanding these precautions, gastro-intestinal irritation should result from the lead, I have always found it easily removed by the prompt administration of a dose or two of castor oil, with the free use of mucilaginous diluents and a farinaceous diet for two or three days.

The diet for aneurism should be as *dry* as is compatible with the patient's comfort; since much liquid tends to fill the vascular system with aqueous blood, which it is the object of the treatment to prevent. To those who have a strong disposition rapidly to reproduce rich, fibrinous blood, animal food should be allowed only sparingly,—for instance, not oftener than every second or third day. But in a large proportion, this disposition does not exist: on the contrary, under a farinaceous or vegetable diet, there is often a decided tendency to an impoverished state of the blood. Here, animal food should be allowed daily. In short, the principle of keeping the patient low, yet just short of that degree which would induce anæmia, should be the practitioner's constant guide.

The utmost corporeal quiescence is indispensable; as acceleration of the circulation by efforts not only defeats the object of the treatment, but is even incompatible with safety, as rupture of the sac might be the consequence. The patient ought, in fact, to be almost constantly in the sitting or lying position. If exercise be permitted at all, it should not exceed a quiet pace about the room, or gestation in an easy carriage, into which the patient should submit to be lifted. On the same principle, the utmost mental tranquillity is desirable.

External applications are not to be neglected under suitable circumstances. When there is much pain in the tumor, leeches sometimes afford great relief; but, when the integuments are very thin and discoloured, they should not be applied to the immediate part, lest they should induce sloughing and rupture of the sac.

Ice, as an application to the tumor, has been strongly recommended; but the pain which it produces is in general intolerable beyond a short time. Its occasional use, however, and, in the intervals, a cold cataplasm of linseed meal and vinegar, are very serviceable by contracting all the tissues, and promoting the coagulation of the blood within the sac, when its current has been rendered languid by depletory measures. When cold applications are not employed, and the tumor is painful and requires support, I have found the emplastrum Belladonnæ afford the greatest relief.

When the aneurism is of the false, or even of the mixed species, as may in general be presumed when it is seated in the descending aorta, whether thoracic or abdominal, the treatment should be steadily pursued for one, two, or three years, with a *curative* object; for experience has proved that such aneurisms occasionally admit of a radical cure. I mention so long a period as three years, because a patient should not venture to return to active habits, until a year at least has elapsed after the disappearance of all the symptoms; such a term being requisite before the coagulum which has filled the sac can undergo a sufficiently firm organization and induration to render the reparation secure. When the aneurism is of the true species, which is generally the case with those of the ascending aorta and arch, a cure is scarcely to be anticipated, as coagula can scarcely ever be made to form in the sac. Still, by a judicious management of the treatment, a valuable life may often be greatly prolonged.

In cases of mere dilatation of the aorta or arch, as the immediate danger is by no means so great, more latitude in exercise may be permitted. The object here is, in the first place, to obviate the increase of the dilatation, and, in the second, to prevent its inducing enlargement of the heart by the obstacle that it presents to the circulation. By the tranquil system which has been pointed out, these two objects may frequently be attained, and the patient's life prolonged for an indefinite series of years.

Treatment of Anæmic and Nervous Pulsation of the Aorta. —If the case be one of mere anæmia, without more nervous excitability than that state ordinarily induces, the patient may at once be submitted to the almost infallible remedies for anæmia; namely, iron in large doses, continued for a month or two; aloetic aperients, sufficient to move the body once or twice daily, without relaxation; and a large proportion of slightly under-dressed animal food, at breakfast and dinner. Wine and porter are too stimulating till the anæmic state is nearly gone. A dry bracing air, much out-door occupation *short of fatigue*, and a cheerful, amused state of the mind, are most desirable auxiliaries. The drain of leucorrhœa, if it exist, must of course be arrested, and this object may in general be easily attained by the daily injection of half a pint of cold water containing ʒss of the liq. plumbi acetatis. The bleeding of piles also demands the immediate use of cold water lavements, or of the other usual remedies for that affection. Menorrhagia should be checked by the ordinary means.

If the patient be more than commonly nervous, the above remedies are apt to prove too stimulant in the first instance, and they should therefore be preceded for a week or two by a broth and fish diet, adequate aperients, sedatives as hyoscyamus, tr. lupuli, extr. lactucæ or conii, and, if there be hysterical symptoms, by these conjoined with antispasmodics, assafœtida, galbanum, valerian, musk, æther, shower-bath, &c. Iron and animal food should then be commenced in moderate doses, and gradually increased.

It is in vain to attempt the latter remedies while there is any chronic gastro-enteritis or colitis in existence,—affections which are a common cause of anæmia, and which require an opposite treatment.

CHAPTER XI.

MALFORMATIONS OF THE HEART.

MALFORMATIONS of the heart are imperfections, generally congenital, in the structure of the organ, and they consist in a deficiency, a superabundance, or an anomalous configuration of parts. The number of varieties of malformation is considerable, and they are so irregular in their combinations as scarcely to admit of being classified on general principles. All worthy of notice that have hitherto been met with, are comprised in the following catalogue.

1. The heart is single, like that of a fish, consisting of one auricle, and one ventricle from which springs a trunk that presently divides into the aorta and pulmonary artery. The patients have generally died within ten days.*

2. There are two auricles, and one ventricle. In one case the patient attained the age of twenty-two.†

3. The foramen ovale remains open. This is the most common malformation, and is found at all ages, sometimes even at the extreme period of senility.‡

4. The foramen ovale and ductus arteriosus both remain open.§

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* Vid. a Case in the Philos. Trans. v. 88, p. 346 ; another, ibid. v. 95, p. 228 ; another in Dr. Farre's Path. Research. Essay 1, p. 2 ; and two in the Ephem. nat. cur. Dec. 1, ann. 4 and 5, Obs. 40 ; and Dec. 2, ann. 10, Obs. 44.

† Case by Wolf, mentioned by Kreysig, die Krankeitendes Hertzens. Berlin, de 1814 à 1817, v. iii. p. 200 ; and one was seen by Breschet.

‡ Passim. It has been found in the aged by Albinus. Academ. Annot. Lib. i. cap. ix.; and Burns on Diseases of the Heart, p. 8.

§ Deschamps, Fouquier, Thibert, Monro, Burns, &c.

5. The foramen ovale and ductus arteriosus are open, and the pulmonary artery obliterated at its origin. In one case, the cavity of the right ventricle was nearly obliterated, and in two others the septum of the ventricles was perforated.[*]

6. The septum of the ventricles is totally deficient, and that of the auricles very imperfect.[†]

7. The aorta arises from both ventricles, i. e. The septum of the ventricles being deficient at the mouth of the aorta, forms a common opening between that vessel and the two ventricles. It is generally accompanied with contraction of the pulmonary artery, frequently with an open state of the foramen ovale, and occasionally with obliteration of the pulmonary artery and patescence of the ductus arteriosus.[‡]

8. The septum of the ventricles is perforated. The aperture is small, and, though near, it is not immediately in, the mouth of the aorta. With this state, the pulmonary artery is sometimes contracted, and the foramen ovale open.[§] A similar perforation appears to be formed by ulceration, and this, in one case, took place at the point of junction of the septum of the auricles and ventricles, so that the four cavities of the heart communicated.[||]

9. The pulmonary artery arises from both ventricles, and the foramen ovale is open. This vessel sends off the *descending* aorta, while the *ascending* arises in the natural way.[¶]

10. The aorta springs from the right ventricle, and the pulmonary artery from the left, the foramen ovale, and sometimes also the ductus arteriosus, remaining open.[**]

11. The right auricle opens into the left ventricle instead of

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[*] W. Hunter, Med. Obs. and Inq. v. vi. p. 291.—Farre, two cases, Path. Research. p. 19. Two died within thirteen days: one lived six months.

[†] Farre, Path. Research. p. 30. Senac, Traité sur la Structure du Cœur, v. ii. p. 404.

[‡] Corvisart, p. 293-8, three cases.—Sandifort, Obs. Anat. Path. cap. 1, p. 35.—Bartholinum, Acta Hofniensia, tom. i. p. 200.—Abernethy, Surg. and Phys. Essays. —Farre, Path. Res. p. 26.—Ed. Med. and Surg. Jour. vol. ix. p. 399. Tredeman, Stander. The Writer, p. 491.

[§] Dr. Hunter, Med. Obs. and Inq. v. vi. p. 299, two cases.—Corvisart, p. 276.

[||] Laennec, tom. ii, p. 547.—Thibert, Bouillaud.

[¶] Two Cases by Sir A. Cooper.

[**] Farre, Path. Research. p. 29.—Langstaff, Lond. Med. Rev. p. 88.—Baillie, Morbid Anat.

into the right, and the ventricles communicate by an aperture immediately below the aortic valves. The foramen ovale is open.*

12. The arch of the aorta was double in a child of twelve or thirteen years old seen by Bertin the father.

13. The foramen ovale is closed in the fœtus.†

14. The valves sometimes exhibit defects which have been supposed to be congenital, but which are more probably referable to endocarditis, if the patient has ever laboured under that affection: namely, the mitral, the tricuspid, and the pulmonic valves have been found stretched flat across their orifices, with a perforation in the centre.‡ The membranous part of the several valves has been found perforated: in one instance it resembled a net-work.§ I have seen several such. They have been supposed to be congenital affections, but they are mere results of atrophy, and occur in the atrophous and anæmic alone.

Of all the causes of communication between the two sides of the heart, patescence of the foramen ovale is the most frequent. This either results from the two layers of which the valve consists in the fœtus, not becoming adherent,—a common form of patescence, and one which does not appear to occasion any material inconvenience: or the foramen is dilated and permanently open, being sometimes large enough to admit the thumb. This dilated state is generally congenital; Louis thinks it is almost always so: but, as many patients have dated their symptoms of disease of the heart from a fall, blow, or violent effort, it is probable that, in such cases, these accidents had caused either the rupture of the membrane closing the foramen, or the separation of its imperfectly agglutinated layers; whence ensued the progressive enlargement of the aperture. Bouillaud gives two cases of communication from ulceration through the partitions.

Whatever be the mode of communication between the two sides of the heart, its effect is, with few exceptions, to cause an intermixture of the arterial and venous blood. One exception, and the most common, is, when the two layers of the foramen ovale are simply non-adherent; for they are then closed like an oblique valve by the pressure of the blood on each side, a pressure which exists as well during the diastole, as the systole of the auricles; for, according to the experiments of the writer, the auricles are constantly *full*, though sometimes more distended than at others. A second exception may possibly exist when the pressure of blood on each side of a gaping aperture is equal. But such cases are, I believe, more imaginary than real; for it scarcely ever happens that there is not, on one side or the other, some valvular or analogous obstruction, which, by impeding the current of the blood along its natural channel, renders its pressure through the morbid aperture stronger than that in the opposite direction. Thus, in more than half the cases of communication between the right and left cavities of the heart, there is a contraction of the pulmonary orifice, or of the pulmonary artery itself, and this, by gorging the right auricle, causes a predominant pressure of blood into the left, when the foramen ovale is open: when it is not, the pulmonic contraction, assisted, as is mostly the case, by hypertrophy of the right ventricle, might even occasion a predominant pressure of blood into the left ventricle through an aperture in the septum. When there is no contraction of the right orifices, the superior strength of the left ventricle would cause the passage of blood out of this cavity, or even out of the aorta (case of Mitchell), through a morbid aperture, into the right ventricle: and a contraction of the aorta or left orifices would occasion a predominant pressure out of the left auricle into the right, supposing the foramen ovale to be open.

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* A case by Holmes, Ed. Med. Chirurg. Trans. p. 252. The right auricle equalled a pint in capacity. The patient attained the age of twenty-one.

† Vieussens sur la Structure du Cœur, c. viii. p. 35.

‡ Burns, Morgagni, Laennec, Louis, Bertin.

§ Laennec, ii. p. 550.

The communication of the two sides of the heart is almost constantly accompanied with hypertrophy or dilatation of the right cavities, whereas the left are very rarely affected. This remark has been corroborated by the subsequent cases of Louis and of Bouillaud. Thus, of twenty cases by Louis, dilatation, six times with hypertrophy, and twice with attenuation, affected the right auricle in nineteen. Dilatation affected the right ventricle in ten; hypertrophy in eleven; hypertrophy with dilatation in five.

Whereas, on the left side of the heart, dilatation of the auricle was observed in three only; that of the ventricle in four; its hypertrophy in three; and hypertrophy of the auricle in two,—precisely the inverse of what is ordinarily seen.

Of eleven cases in which the size of the heart was noticed by Bouillaud, dilatation of the right auricle existed in ten,—five times with hypertrophy: hypertrophy of the right ventricle existed in ten, four times with contraction. The left cavities presented nothing particular, except in three cases, in which there was valvular contraction of the left orifices (Traité, ii. p. 567).

M. M. Bertin and Bouillaud attribute the hypertrophy to the introduction of a certain quantity of red, *arterialised* blood into the right cavities, which they think calculated to occasion their hypertrophy in consequence of its being more irritating, more nutritive, possessed of more vitality, than the venous blood.

I doubt whether this ingenious hypothesis is tenable, as the most remarkable cases of hypertrophy of the right ventricle have been those in which there was extreme contraction of the pulmonary orifice, when, consequently, the current through the foramen ovale must have been so decidedly from the right to the left side, that no arterial blood could possibly have entered the right ventricle.

What, then, was the cause of the hypertrophy of that ventricle? The contraction, I should imagine, of its pulmonary orifice; in the same way that contraction of the aortic orifice occasions hypertrophy of the left ventricle. M. M. Bertin and Bouillaud support their opinion by the circumstance that the hypertrophy is often accompanied with contraction of the cavity, which, they think, would not be the case if the hypertrophy resulted merely from too great a quantity or too great a distending pressure of the blood. To this it may be replied that, in the left ventricle, hypertrophy with contraction arises more frequently from a similar cause, that is to say, obstruction of the aortic orifice, than from any other; the reason of which I have attempted to explain in the article on hypertrophy, p. 250. It might be objected to this, that, in many cases, though the pulmonary artery was obstructed, the ventricle discharged itself by an opening into the left ventricle or into the aorta. True; but this discharge was not made with the same facility as in the natural way through the pulmonary artery, inasmuch as the weight of the aortic circulation exceeds that of the pulmonary.

Having said so much to account for the hypertrophy, we have next to consider the cause of the dilatation which is occasionally found in the right cavities. This is manifestly an effect of over-distention; for, as far as I can discover, it is always accompanied with an excess of blood gorging the right cavities, in consequence of a mechanical obstacle, or impediment in front of the cavity dilated. Thus, in a case by Corvisart (p. 279), the excessive smallness of the aorta caused the blood to flow out of the left auricle into the right, through the foramen ovale, which was more than an inch in diameter, and thus to produce dilatation with hypertrophy of the right cavities. So, again, the right ventricle is apt to become dilated when the weight of the aortic circulation is thrown upon it by a communication between the two ventricles.

While I thus contend that there are sufficient *mechanical* causes to account for hypertrophy and dilatation of the right cavities in cases of communication between the two sides, I do not wish to assert that the introduction of arterial blood may not contribute to the production of hypertrophy. On the contrary, I think it probable that it does, since the arterial blood is a morbid stimulus of the right cavities; but it is repugnant to the principles of inductive science to assign this, which is at best problematical, as the sole cause, overlooking others, the effect of which is unquestionable. M. Bouillaud, in his recent work, has adopted the same view (ii. p. 575).

General signs of communication between the two sides of the heart.—The signs given by authors, are, a violet or blue colour of the skin, in general much more intense and extensive than in any other malady, and sometimes even universal; a reduction of temperature, with great sensibility to cold; unusually frequent attacks of syncope; occasionally, convulsions; and a greater difficulty of the respiration than in most other diseases of the heart.

These symptoms are sufficiently correct in reference to the cases in which there is distinctly a violet or blue tint, with its almost inseparable concomitant, an obstructed circulation; but there are numerous cases of communication, in which the intermixture of venous and arterial blood, and the obstruction of the circulation, are so inconsiderable, that the blue tint is absent, and the general signs are only those of a moderate valvular obstruction. Here, the physical signs and the history are the only means by which we can arrive at the diagnosis, and it will be convenient to consider them conjointly under the head of physical signs, to which we shall presently come. Meanwhile, it is necessary to revert to the blue discoloration of the skin, (designated by the names *blue disease*, *blue jaundice*, *cyanosis*,) as its causes and circumstances do not appear to me to have been fully understood and explained by authors. When the intermixture of the arterial and venous blood is not very considerable, and especially when the *admission of venous blood into the lungs is free*, the discoloration is sometimes not deeper than is to be found in cases of ordinary obstruction to the return of the venous blood, and occasionally it scarcely exists at all. On the contrary, when the ingress of venous blood into the lungs is very limited, and the intermixture with the arterial considerable, the colour is of the deepest dye, and pervades not only the lips, nose, ears, and face, but the hands, the feet, and, in greater or less intensity, the skin universally. Such, at least, is the generalization to which I have been brought by the cases that I have seen, and by an examination of nearly all that have been published on this subject. M. Jul. Cloquet and M. Bouillaud say that when red blood passes from the left into the right cavities, it cannot occasion cyanosis (Traité de Bouillaud, ii. p. 575 and 573); but this is a mistake; for, if arterial blood replace venous in the right cavities, a diminished quantity of venous blood is transmitted to the lungs for arterialization; whence the total mass in the system is darkened. This occurred, for instance, in the case of Mitchell, whose tint was unusually dark.

According to M. Laennec, the blue colour of the skin is equally marked and extensive in some diseases of the lungs, particularly emphysema, as in cases of communication between the two sides of the heart. This is not consistent with my own observation; for, of many thousand cases of pulmonary disease which I have seen, in not one, nor in any ordinary organic disease of the heart, has the colour admitted of comparison with that which I have witnessed, and of which I shall presently offer an instance, in cases of the communication in question.

For this reason I must dissent from the opinion of M. M. Bertin and Bouillaud, and of M. Bouillaud in his later work, who maintain that the blue or violet colour depends, not on intermixture of the black with the florid blood, but "principally, if not exclusively," on the same cause that occasions it in cases of ordinary obstruction to the circulation: namely, "the stagnation of the blood in the right cavities of the heart, and in the venous system, which is as it were gorged with it." M. M. Louis and Ferrus entertain the same opinion. Were this true, cases of *intense* discoloration would be of ordinary occurrence, instead of being extremely rare, and presenting themselves in those almost exclusively, who are affected with a communication between the two sides of the heart.

Venous retardation, however, co-operates with the intermixture of blood in darkening the colour, and it is also the main cause of dropsy, passive hæmorrhages, &c.

It is scarcely necessary to remark, that, when hypertrophy or dilatation co-exists with malformation, the effects and signs resulting from them are added to those of the congenital disease.

Physical Signs.—Laennec had not an opportunity of studying cases of malformation with the stethoscope. In a case subjoined (Collins), of which I made a post-mortem examination, the signs were conformable to the general principles developed in the several articles on organic diseases of the heart: namely, enlargement of the organ by one-half was indicated by dulness of the præcordial region on percussion and slight prominence; hypertrophy of the right ventricle was denoted by increased impulse at the inferior part of the sternum; contraction of the pulmonary orifice, and a common opening of the right ventricle with the left into the aorta, occasioned a loud, *superficial* hissing murmur with the first sound, loudest about the middle of the sternum, over the orifices affected.

In another case subjoined, of a living patient (Master R.), the signs were almost identical. In a third, a young lady, æt. 12, with cyanosis, there was an exceedingly loud and *superficial* murmur over the pulmonary artery, from its valves to the top of the sternum. I have met with a few other cases presenting more or less similar signs, but have not had the opportunity of post-mortem verification, except in the single case of Collins. The cases of other authors afford little or no information respecting the physical signs, as murmurs are very rarely mentioned, and when they are, it is only in general terms. Thus, out of fifteen cases, collected from various sources by Bouillaud, in one only is it stated that there was a bruit de soufflet in the præcordial region. Drawing, therefore, from my own limited observations until a greater number of cases have been carefully collected, and guided by the general principles which are offered in this work as applicable to particular valvular diagnosis, I should think that the murmurs indicative of a communication between the two sides of the heart would be nearly as follows:—

An unusually loud and *superficial* or *near sounding* murmur with the first sound, *immediately* over the semilunar valves, (i. e. about opposite to the inferior margin of the third rib,) is generally seated in the mouth of the right ventricle, and may proceed either from a contraction of the pulmonic valves or orifice, or from an opening out of the right into the left ventricle, or from both these lesions conjoined. If it proceed from contraction of the pulmonic valves or orifice alone, it will be audible along the course of the pulmonary artery, up to the second intercostal space, much more distinctly than along the course of the aorta, and will be attended with a thrill. If it proceed solely from an opening out of the mouth of the right into the left ventricle, (the pulmonic orifice being either healthy or totally obliterated,) it will be more audible along the course of the aorta than along that of the pulmonary artery. If it proceed from the double lesion, viz. a contracted pulmonic orifice and an opening into the left ventricle, it will be loudly audible along the course of both vessels, and a thrill will be felt over the pulmonary artery. When these signs of a lesion in the mouth of the right ventricle coincide with cyanosis, the evidence of a communication between the two sides of the heart is almost positive, and, as hypertrophy of the right ventricle is usually a concomitant, its presence is a corroborative circumstance. When the signs in question do *not* coincide with cyanosis, an appeal must be made to the history of the case. If it appear that the patient has exhibited the symptoms of organic disease of the heart from early infancy, yet has never been affected with endocarditis to which the valvular disease could be ascribed, there are strong probabilities of a congenital malformation, and presumptions of a communication between the two sides,—though without so considerable an intermixture of blood, or so great an obstacle to its ingress into the lungs, as suffices to occasion cyanosis.

A further appeal may be made to numerical considerations. The frequency of contraction of the right orifices, especially the pulmonic, in cases of communication of the two sides of the heart, is very striking. Of 50 cases of cyanosis collected by M. Gintrac, 27 presented obstruction of orifices, and its seat in 26 of them was in the pulmonic orifice, and in 1 in the tricuspid. Of 15 cases collected by Bouillaud, 12 presented valvular disease; in 10 of which there was contraction of the orifice. In 8 out of the 12, the lesion was in the right valves; and in 5 out of these 8, the pulmonic valves were its seat, in 1 the pulmonic and tricuspid together, and in 2 the tricuspid alone. Thus, in cyanosis, the numerical chances, according to the above cases, are 32 to 3 or more than 10 to 1, that there will be contraction of the pulmonic orifice: and conversely, as contraction of this orifice from ordinary causes is extremely rare, its presence, when not distinctly traceable to inflammation, affords very strong numerical presumptions that there is a communication between the two sides of the heart, even though cyanosis be absent.

Against one source of fallacy, the auscultator must be on his guard: namely, that of mistaking a dilatation of the pulmonary artery for a lesion in the mouth of the right ventricle. In the latter case, the murmur will be loudest immediately over the valves: in the former, it will be loudest at the second intercostal space, where a strong thrill and pulsation will also be perceptible (See cases of Weatherly and L. P.).

Cases.—The two following cases present excellent exemplifications of cyanosis. The history of the latter was drawn up, at my request, by the father, a gentleman of great intelligence and observation. It is valuable as displaying the habitudes of a patient affected with this disease in a much more graphic manner than can be done by a mere enumeration of symptoms.

Mary Collins, æt. 8, applied to me October 22d, 1830. Lips, nose, cheeks, palpebræ, hands and feet, of a violet colour: tongue and mouth still darker. On a frosty day, after walking or ascending stairs, the hue of the parts enumerated, as witnessed by myself and several medical friends, is equal to the deep stain communicated to the skin by black currants or the small black cherry, and the face and hands universally are as dark as those of a mulatto. Children in the streets often inquire in winter, "where she got blackberries at that season." Dyspnœa on the slightest exertion, particularly ascending; cough when hurried, not otherwise: sternum very prominent; great sensibility to cold —constantly steals to the fire, even in summer; headache, vertigo, drowsiness, and sluggishness. Pulse very small and weak, and when hurried, it is irregular, intermittent and unequal.

Auscultation.—Resonance of the præcordial region dull. *Impulse,* considerably stronger and more extensive than natural—strongest over the right ventricle. *Sounds.* The first, a very loud, hissing, superficial bellows-murmur: the second, natural or nearly so. In six months she died of a chronic abscess in the brain.

Diagnosis. Patescence of the foramen ovale; hypertrophy of the right ventricle; obstruction of the orifice of the pulmonary artery. (Stated before the dissection, at which Dr. Marshal Hall, Mr. Else, surgeon, and others were present.)

Sectio.—The heart was one-half larger than natural: the walls of the right ventricle were thickened to half an inch: the cavity was slightly dilated and its pulmonary orifice contracted to the size of a goose-quill, while a common opening from the right ventricle, admitting the index finger, existed into the aorta and the left ventricle. The left ventricle was one-third of an inch thick, and its cavity about natural. The two layers of the foramen ovale were disunited, and the handle of a large scalpel easily passed obliquely through them. The lungs were rather flaccid, imperfectly crepitant, and universally gorged with black blood.

In this case, it was the *superficial* nature of the murmur which led me to conceive that it was occasioned by an obstacle to the passage of blood from the right, rather than from the left ventricle.

Master R. æt. 11. Consulted me in 1830. While he is tranquil and warm, the complexion is about two shades darker than natural, and its tint is a purplish crimson: on the lips the colour is deeper, and within the mouth it is a blackish violet. On ascending a flight of stairs, the colours become intense. The hands and feet are of the same hue, and the last phalanges of the fingers and toes are bulbous, being one-third larger in circumference than the phalanges above. Pulsation of the carotids: engorgement, without pulsation, of all the veins of the neck. Left margin of the sternum prominent opposite to the fifth, sixth, and seventh ribs. *Resonance* deficient over the whole præcordial region. *Impulse* much increased, strongest where the sternum is prominent. *Sounds.*—The first is a loud, prolonged, bellows-murmur, loudest opposite to the arterial orifices of the heart: the second is short and loud, with a very slight filing murmur.

The following history is given by the father. "He was always as blue as at present, and I think more so when very young. Until the age of nine months, he was very subject to spasms of the bowels, and also, as I used to think, of the chest. They were in general relieved by immersion of the lower extremities in warm water, (which was always kept so as to be ready within two minutes,) and by a dose of castor oil in anise water. His respiration was always quick, and he was always subject to cough, particularly on taking cold, to which he is very liable. He perspires very freely, and, about the hands and feet, to a great degree. When the weather is sharp and nipping, he becomes exceedingly blue. He is very chilly, and sensible to cold. When exposed to a cold or damp and cold atmosphere, he becomes as it were *asthmatic*; his corporeal powers are overcome with numbness, he loses, in a great measure, the faculty of motion, and I am sure that, if placed, on a cold day, particularly with an east wind, in an exposed situation two miles from home, and left to return that distance by his own exertions, his powers would become so deadened that he would perish in the attempt. I have sometimes, under such circumstances, been obliged to bring him home in a coach, or even in my arms. He suffers less in severe *frosty* weather, than when the wind blows cold and harsh, though the temperature be six or eight degrees above the freezing point. However much inconvenienced by exposure to cold, he regains all his powers by the time that he has remained half an hour in a warm room. In mild weather he is less blue, and his respiration less oppressed. If he cuts or scratches himself, he bleeds more than others. The finger and toe-nails are scarcely thicker than paper, and they grow very fast, requiring to be cut every four or five days. He is very subject to cynanche tonsillaris, with great swelling of the parts. He suffers more than other children from illness produced by slight or common causes, and his health consequently suffers frequent interruptions. I must remark, in particular, that when he becomes ill, it is not gradually, by the progressive developement of the symptoms, but suddenly, scarcely ever giving the slightest premonitory signs. Not half an hour before a severe attack, he has often appeared in good health. He grows remarkably fast. His temper is very quick and irritable, but his disposition is candid, frank and generous: his mind is active and ardent. Tongue never quite clean, and the papillæ are very large: appetite and digestion generally good: bowels free: urine almost always turbid."

I have lost sight of the patient since the above was written eight years ago.

PART IV.

NERVOUS AFFECTIONS OF THE HEART.

The nerves of the heart, as of every other organ, may be affected in two ways. They may labour under over-excitement, dependent either on increased irritability or on excessive stimulation; and they may be in a state of deficient excitement, dependent either on diminished irritability or on inadequate stimulation. These states, when existing in a moderate degree, cannot strictly be considered morbid. Thus, palpitation from exercise or from an exhilarating mental emotion, and languor of the heart's action from a depressing passion, do not rank as diseases. But when the states in question exist in excess, and when they result, less from remote sympathies, than from a primitive affection of the nerves of the heart itself, they constitute diseases. Of these, the state of over-excitement comprises *Neuralgia of the heart, or Angina pectoris* and *Palpitation*; while the state of deficient excitement presents *Syncope*. These diseases will be considered in successive chapters.

Spasm of the heart, a disease imagined by Laennec alone, I believe to be really imaginary; for I have shown that the murmurs of the heart and arteries, independent of organic disease, which he adduced as its sole proofs, are dependent on other well-defined causes (See p. 95). *Convulsions* of the heart, if not also imaginary, do not admit of proof. *Paralysis* may result from tobacco and some corrosive and irritant poisons; but it is foreign to the subject of this work.

CHAPTER I.

NEURALGIA OF THE HEART, OR ANGINA PECTORIS.

Neuralgia of the heart occurs in paroxysms of greater or less severity, and at longer or shorter intervals. When presenting the train of symptoms which have been denominated by Dr. Heberden *angina pectoris*, it commences by a sensation of pain and constriction in the præcordial region, accompanied with a more or less painful numbness in the left arm, more rarely in both arms, still more rarely in the right arm alone, and occasionally in all four extremities, of which I have seen several instances.

At first the pain may not reach beyond the insertion of the deltoid muscle, but it soon extends down the inside of the arm to the elbow, and sometimes accompanies the ulnar nerve to the extremities of the fingers. It is not unusual for pain to exist at the same time in the left anterior part of the chest, following the tract of the anterior thoracic nerves. In females, it is often attended with extensive cutaneous neuralgia, rendering the mammæ so sensitive, that the slightest pressure becomes painful. When the attack is smart or what is called *acute*, the pain in the heart is excruciating, appearing to the patient as if "iron nails or the claw of an animal tore asunder the anterior part of his chest" (Laennec). With this, there is great pulmonary oppression, amounting, in the worst cases, to suffocative orthopnœa; the heart either palpitates violently, or it falters, flutters and intermits:* congestion of blood in the head, syncope and convulsions sometimes ensue.

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* I suspect that some authors, who have described the pulse as calm, have mistaken these characters for calmness. I have never seen the action of the heart undisturbed in a *severe* case, nor do I think it possible; but it is often so in slight cases.

The attack is commonly induced by some over-excitement of the heart, especially that of walking up hill, to the effect of which a recently loaded stomach and a wind in front, powerfully contribute, —the latter appearing to act by stimulating the excito-motory nerves of the face. The patient is compelled instantly to stop, and, if the complaint be recent, the attack sometimes subsides in the course of a few minutes by mere rest, and seldom continues longer than from half an hour to an hour, even in cases so severe as to prove fatal. The disease is apt to become chronic;—when it recurs more frequently, is excited by slighter causes, and is sometimes so obstinate as to resist every remedy for several hours, and even for as many days. In these cases it is seldom that there are not some of the physical as well as general signs denoting organic disease of the heart, and that the latter is not found on post-mortem examination.

Causes of angina pectoris.—Great diversity of opinion has existed respecting the cause of angina pectoris. Different physicians have found it connected with different organic lesions or states, and each has supposed it to be occasioned by that, with which he has most freqnently found it co-exist. Dr. Parry, and after him Burns and Kreysig, ascribe it to ossification of the coronary arteries; Dr. Hooper, to affections of the pericardium; Dr. Hosack, to plethora; Dr. Darwin, to asthmatic cramp of the diaphragm; Drs. Butler, Macqueen, and many others, have regarded it as a particular species of gout; Dr. Latham has found it connected with enlargements of the abdominal viscera, while the thoracic viscera were sound; and Heberden, having found it both connected and unconnected with organic disease, thinks that its cause has not been traced out, but that it does not seem to originate *necessarily* in any structural derangement of the organ affected.

They who have ascribed angina pectoris to any particular cause to the exclusion of others, have unquestionably taken too limited a view of the subject; as experience has fully proved that it may originate in various causes. According to my own observation, it may originate in any cause, whether organic or functional, capable of *irritating* the heart, or of rendering it morbidly susceptible of irritation; and as structural disease of the organ has this effect more than other cause, it is that on which the malady, in its severer forms, is most frequently dependent.

The most violent cases of angina that have occurred to myself, and, if I mistake not, that have been recorded in books, have been connected with osseous, cartilaginous, steatomatous or other degenerations of the heart or great vessels, by which some portion of them, especially the coronary arteries, the valves and the commencement of the aorta, was more or less deprived of its elasticity. Hence it may be perhaps reasonably conjectured that, when the action of the organ is excited, as by ascending a hill, a loaded stomach, &c. the over-tension of the rigid portion is the source of the irritation and pain. It is no objection to this view that, at the time when the pain is the most intense, the action of the heart is sometimes diminished—that it feebly flutters and falters, and that the pulse has the same characters; for, so long as these phenomena display themselves, the heart is in a state of engorgement—of even greater distention, perhaps, than when it is acting violently. The proofs that such is the case are manifest in the suffocative orthopnœa, the tumid, livid state of the face, and the diminution of the sounds of the heart.

I have also several times seen angina of considerable, but not equal severity, accompany hypertrophy and dilatation with or without softening; but I have never known the malady to exist *in an aggravated form*—one which truly merited the name of *angina* rather than of mere neuralgia, independent of *some* organic disease of the heart or its immediate appendages. Cases, it is true, are on record which appear to militate against this opinion; but as it is only of late years that the anatomical characters of hypertrophy, of dilatation, and of softening have been clearly understood, the evidence of such cases must be admitted with reserve. To mention an instance,—a case of sudden death from angina, said to be independent of disease of the heart, was recently communicated to me; but, from the statements of the parties present at the dissection, it was clear that there existed a marked dilatation, which they had not recognized.

Angina, however, in a moderate degree may, as Laennec maintains, exist independent of any organic disease of the heart or great vessels, and it is, indeed, a very common affection. I have frequently met with it in nervous or hysterical females subject to palpitation, in nervous males, in cases of nervous dyspepsia and hypochondriasis, and in mere plethora. It occurs in these cases under the form of occasional aching pains in the anterior part of the chest, extending sometimes to the neck and stomach, and attended or not with pain and numbness in one or both arms. One of my medical friends always feels the affection of the arm when attacked with dyspeptic palpitation, to which he is subject.

It is very conceivable that, if the irritation of a loaded and dyspeptic stomach can create angina, an enlarged liver or other abdominal tumor, by displacing the diaphragm, or by mere sympathetic irritation, might, as in the cases of Dr. Latham, produce the same effect.

Nerves affected in Angina.—M. Desportes places the seat of angina in the pneumogastric nerve or par vagum, because the lungs, as well as the heart, are affected with pain and have their function disturbed. Laennec thinks that the filaments which the heart derives from the sympathetic, are likewise implicated in the disease; because there is sometimes pain in the organ without any in the lungs or material embarrassment of the respiration. M. Bouillaud thinks that, as the healthy heart does not appear to enjoy any *animal sensibility* (Bichat), the *pain* of angina is seated in the phrenic and intercostal nerves; whereas, the nerves of the heart itself, simultaneously affected, reveal their morbid condition, not by pain, but by disturbances in the movements of the organ, accompanied with that internal, undefinable uneasiness which precedes faintness or syncope. Whatever be the nerves in which the pain is seated, it is propagated, either by sympathy or by anastomosis, to others: namely, to the superficial cervical plexus and its anterior thoracic branches, whence proceeds the pain in the neck and on the surface of the chest; to the branches of the brachial plexus, especially the ulnar, whence arises the pain descending to the elbow and sometimes to the fingers; finally, to the branches of the lumbar and sacral plexus, whence the pain and numbness felt in the thighs and legs, and even in the spermatic cord and testicles.

The nature and variability of the symptoms of angina pectoris confirm the opinion of Laennec that it is a neuralgic affection;

for those neuralgic affections whose nature is least equivocal,—sciatica or tic douloureux, for instance, produce, in different degrees, effects of the same nature and equally diversified as those of angina pectoris; that is to say, acute pain, painful torpor, simple numbness in the tract of the affected nerve, and sometimes spasm and sub-inflammatory intumescence of the parts to which the nerve is distributed. I have known malaria produce intermittent, periodic neuralgia, not only in every extremity, but also in the heart.

Diagnosis.—The point of importance is, to ascertain whether there be disease of the heart, and this is to be done by the signs fully explained in this work.

Prognosis.—When the malady is dependent on organic disease of the valves, or of the great vessels, the prognosis is decidedly unfavourable; for in addition to the danger which always attends the organic diseases in question, there is that of fatal syncope from the angina—a termination to which this affection is prone. When angina is connected with hypertrophy, or dilatation, I have generally found it curable by the means prescribed for the latter diseases. When the complaint is symptomatic of dyspepsia, hysteria, plethora, &c., the prognosis is favourable.

Treatment.—When angina depends on organic disease of the heart, it must be treated on the general principles which regulate the treatment of the latter. The patient should instantly be placed in a state of repose; flatus of the stomach, if present, should be extricated by a draught of peppermint-water with anise oil, sp. ætheris sulph. comp. and aromatic confection: acidity should be neutralised by a free dose of soda or prepared chalk; and if the stomach be loaded with an irritating mass of undigested food, it should be evacuated by ipecacuan with sulphate of copper or of zinc, provided the state of the respiration be such as to admit of the effort of vomiting. Should the distress be extreme and the patient plethoric, six, eight, or ten ounces of blood may be drawn, either by venesection, or by cupping or leeching on the præcordial region.

These preliminary measures having been carried into effect as expeditiously as possible, an antispasmodic and sedative draught should be administered. It may comprise a full dose of tinct. or extr. opii, or, as less exciting, of the liquor opii sedativus, or of acetate of morphia, with sp. ætheris sulph. comp. ʒss. and mist. camph. or solut. assafœtid. ʒx. The draught and the other measures must be repeated according to existing circumstances, of which the practitioner is the only judge.

During the intervals of the fits, the general health must be improved and the recurrence of the fit prevented by the same means as in organic disease of the heart.

When the complaint is chronic and the pain pretty constant, counter-irritants and derivatives, as blisters, setons, or issues on the præcordial region, have been found useful. The emplast. belladonnæ is also a valuable auxiliary.

In cases dependent on hysteria, dyspepsia, anæmia, &c. the primary malady demands the first attention, while the neuralgic pain may be combated by counter-irritants and occasional sedatives. The metallic tonics, especially the sesquioxyd of iron in doses of from ℈ij. to 1½ thrice a day, and the M. Ferri C. in doses of ℥iss, are the most efficacious internal remedies for it. They act mainly by removing anæmia, which frequently complicates, and even occasions, the hysteria, dyspepsia, nervousness and palpitation. Aloetic aperients and animal food should be prescribed with the iron. Quina is the specific for intermittent angina.

In angina dependent on plethora, bleeding and a restricted, dry diet are the essential remedies. Dyspeptic or nervous symptoms must be treated on the usual principles. (See Palpitation.)

The remedy by which Laennec states that he has most frequently succeeded in procuring alleviation in cases of angina pectoris, and of neuralgia of the heart of a slighter kind and without radiating pain, is the magnet, which he employs in the following manner: he applies two steel plates strongly magnetized, of a line in thickness, of an oval form, and slightly arched so as to apply closely to the thoracic walls, the one on the left præcordial region, and the other on the opposite part of the back, in such a manner that the poles may be exactly opposite and the magnetic current may traverse the part affected. This remedy, Laennec adds, is fallible no less than all those by which we ordinarily combat nervous affections; but it has succeeded in his hands oftener and to a greater extent than any other. When it procures little alleviation in angina, more may sometimes be obtained by applying a small blister under the anterior plate. It should be remarked that they who witnessed the application of the magnet by Laennec, did not, in general, form so favourable an opinion of its utility as that author himself. It appeared to be more successful when combined with acupunctuation. I have not tried either, as they are calculated merely to alleviate the symptom, rather than to cure the disease.

CHAPTER II.

PALPITATION, PARTICULARLY NERVOUS.

After presenting a general view of the nature and causes of palpitation, and adverting briefly to its varieties, I shall dwell more particularly on palpitation from inorganic causes, usually called nervous.

As palpitation is, under all circumstances, dependent on over excitement of the nerves of the heart, the phenomenon, in its essential nature, is always the same. The varieties which it presents arise merely from differences in their causes, and from the different routes which these causes pursue in order to arrive at and convey their stimulus to the heart.

Thus, 1. the blood conveys the stimulus *directly*, and in three ways: *a*. By arriving in excess, as from violent exercise, plethora, &c. I conceive that palpitation from excessive loss of blood and all other forms of anæmia or chlorosis, comes under this head; for, though the quantity of blood be diminished, its remarkable attenuation enables it to traverse the vessels with greater facility; and it probably, therefore, arrives at the heart either in redundant quantity or with morbid velocity. This view is more consistent than the anomalous one of palpitation being produced, in cases of loss of blood and anæmia in general, by a deficiency of the natural stimulus: an explanation which some have offered. As anæmia always increases the nervous irritability, this no doubt co-operates in producing anæmic palpitation. *b*. By gorging the heart, in consequence of its transmission being impeded by an organic disease of the organ, or an obstacle in some other part of the circulation. *c*. By being of too stimulant a nature, in consequence of the diet being exciting.

2. The nerves, on the contrary, convey the stimulus to the cardiac plexus *indirectly*, as is the case in emotions of the mind, in dyspepsia, in hysteria, &c. When the above two classes of causes co-exist, the nerves and the blood may convey the stimulus conjointly.

Palpitation in general may be defined to be an increase in either the force or the frequency, or in both the force and frequency, of the heart's contractions, by which they become not only perceptible, but sometimes very troublesome to the patient. They may vary in force from a scarcely sensible degree, to a violence which is extreme. Not unfrequently the sound of the beats is audible to the patient, especially when lying on his side; and, in this position, the second as well as the first sound may occasionally be perceived.

When the circulation is simply accelerated, as by exercise, &c. in a healthy subject, the palpitation consists in an increase both of the force and the frequency of the heart's action. The same occurs in hypertrophy, and hypertrophy with dilatation. In dilatation with attenuation, palpitation sometimes consists in an increase of the frequency, but not of the strength of the beats, though the patient may experience the sensation of an increased impulse. Palpitation of this kind is sometimes very obstinate. Laennec cites an instance in which it lasted eight days, the pulse constantly beating 160 to 180 per minute. I have found the same remarks apply equally to softening.

It must be recollected that, in every organic disease of the heart, when palpitation becomes extremely violent and prolonged, both the impulse and the sounds may be diminished:—in other words, the heart becomes gorged and incapable of adequately contracting on its contents, sometimes yielding a struggling convulsive impulse, with little sound and a feeble pulse, and, in an ulterior degree, especially during dissolution, scarcely producing either impulse, sound, or pulse. Suffocative dyspnœa, lividity, and extreme distress are always concomitant symptoms.

Palpitation from inorganic causes, usually called nervous, and imitating disease of the heart.—There are few affections which excite more alarm and anxiety in the mind of the patient than this. He fancies himself doomed to become a martyr to organic disease of the heart, of the horrors of which he has an exaggerated idea; and it is the more difficult to divest him of this impression, because the nervous state which gives rise to his complaint, imparts a fanciful, gloomy and desponding tone to his imagination. Members of the medical profession are more apt than others to give way to these feelings; partly from their apprehensions being more keen, and partly from an impression too widely prevalent, that there is difficulty in distinguishing inorganic from organic palpitation, and, consequently, that they must remain in a state of anxious uncertainty. It may be said for the consolation of such, that the diagnosis presents no difficulty to one who, to general signs, adds a knowledge of these afforded by auscultation and percussion. I repeat this opinion with increased confidence in the present edition, not only on the grounds of additional experience, but because the signs both of organic and inorganic disease will now be found much more precise and simple, in consequence of the new lights thrown on particular valvular diagnosis and on inorganic murmurs.

Inorganic palpitation presents certain varieties, which it is of the greatest practical importance to distinguish, as the treatment is different, and even opposite. It may be premised that, in all the varieties, the palpitation will, cæteris paribus, be greater in proportion as the patient is constitutionally of a more nervous, irritable temperament.

1. Palpitation dependent on dyspepsia, hypochondriasis, hysteria, latent gout, mental perturbations either of the exciting or depressing kind, excessive study with deficient sleep, and venereal excesses, constitutes the first variety, and forms a large class. When from these causes, it presents various degrees and characters. The slightest degree of it I should describe, from having occasionally experienced it, to be a tumbling or rolling motion of the heart, with a momentary feeling of tightness and oppression. It is referable to an intermission of the heart's action. In a further degree, as Abercrombie has well described, there is a series of quick, weak, fluttering, irregular beats, with slight anxiety, acceleration of the respiration, and a quivering sensation in the epigastrium: this may last from a few minutes to half an hour or an hour, and occur only at distant and irregular intervals, or repeatedly during the day, especially when the patient is startled. The next degree amounts to a perfect fit of palpitation

consisting in increased impulse, sound and frequency of the beats, sometimes with irregularity, and generally with more or less anxiety, dyspnœa, and even orthopnœa. The attack may be only occasional, or may occur several times a day, or may even last with little intermission for several days together.

The palpitation in question may be distinguished from that of disease of the heart by the palpitation occurring only occasionally: by its not being excited, but, on the contrary, relieved by corporeal exercise of such a nature as would certainly disturb the action of a diseased heart: by its disposition to supervene while the patient is at rest, especially at the commencement of the night when he lies wakeful in bed; by a fluttering in the epigastrium; by the general prevalence of nervous symptoms; by the affection being aggravated when the nervous symptoms undergo an exacerbation; by the pulse and the action of the heart being natural during the intervals between the attacks; and by the absence of valvular and aortic murmurs, and of undue impulse; "the shock, even when it at first appears strong, having little real impulsive force; for it does not sensibly elevate the head of the observer" (Laennec).

To this category some would add, an increase of the palpitation after meals, or when the stomach is deranged, and amelioration produced by dyspeptic remedies; but, as the stomach produces the same effects when there is disease of the heart, these signs are not pathognomonic of nervous palpitation. To this point I would particularly direct the attention of practitioners; because many, in forming their diagnosis of the affections in question, regard the dyspeptic signs as paramount in value to all others, and are apt to refer to the stomach the palpitation which really belongs to organic disease of the heart.

Though the present variety of palpitation is often attended with various familiar nervous affections of the head, as pain or sensations of heat or of cold confined to particular parts and coming and going suddenly, temporary vertigo, tinnitus, and confusion of the sight, not increased by lying or stooping; it is not, when purely nervous and the patient not plethoric, accompanied with genuine signs of cerebral determination or congestion: there is no universal, throbbing headache with weight and tension, increased by stooping or the recumbent position:

no stunning sounds and pains in the head on suddenly lying down or rising up: no permanent somnolency, apoplectic stupor, or regular apoplectic fits, as in hypertrophy, &c.

When it has been ascertained that the palpitation in question is independent of organic disease, the treatment presents no unusual difficulty, and is to be adapted to the nature of the exciting causes specified at the head of this division. It would be foreign to the subject of this work to dwell upon the particular remedial measures.

2. Palpitation from anæmia. I use this as a generic term, applicable to both sexes and to all circumstances, instead of the specific term *chlorosis*, which, before anæmia was understood as a general disease, was applied to females with amenorrhœa, under a twofold error; first, that the catamenial deficiency was the cause of the chlorosis, whereas it is most commonly the effect; secondly, that the complexion was a greenish yellow (χλωρὸς); but I have assured myself by particular observations on upwards of 1000 cases, that the tint in question is nothing more than the residuary colour of the skin when the pink has been withdrawn by anæmia; that the hue is more yellow or *sallow* in females with dark complexions, and *vice versâ*; and that it is equally observable in males. This explanation is offered, because some writers still treat of anæmia and chlorosis as essentially different diseases. In my opinion, there is no difference but in the cause, and it is proper to state that such is the import which I attach to the term anæmia throughout this work. Its causes may be, undue loss of blood in any way, and whether sudden or gradual; draining diseases of any kind; deficient food, especially animal; bad air; chronic diseases of the alimentary canal, lungs or heart; depressing passions; excessive intellectual or corporeal fatigue: in short, anything calculated to deteriorate the general health and impair the functions of assimilation and sanguification.

The connexion between anæmia and affections of the heart and great vessels was not noticed by Corvisart, Laennec, Bertin and Bouillaud, Elliotson, or authors in general. In the first edition of this work, it was shown by the experiments on dogs described above at p. 100, that anæmia was not only a cause of palpitation, but also of the inorganic murmurs of the heart and arteries ascribed by Laennec to spasm, and that it existed in almost all the cases in which Laennec described these murmurs to occur. The palpitation which it occasioned constituted the main grounds on which I objected to the treatment of Valsalva and Albertini for hypertrophy, and substituted another treatment on a less active depleting system. M. Bouillaud, as has been shown above, (p. 123,) has, in his more recent work, followed up the same investigation and arrived at the same conclusions respecting anæmic palpitation. It is, in fact, the variety which, when misunderstood, is by far the most liable to be confounded with organic disease of the heart.

The *general symptoms of anæmia* may be rapidly sketched as follows.—The complexion is unusually, and sometimes singularly, pallid or exsanguine; the lips, the interior of the mouth, and the inside of the palpebræ, partake more or less of the same paleness; the pulse is quick, small, weak and *jerking*, (the pulse of unfilled arteries,) and during palpitation it often presents a thrill; its average frequency is generally above 80 or 90, and under excitement it is easily raised to 120 or 130, and occasionally even to 140 and 150: the slightest causes, including all corporeal efforts, suffice to induce palpitation, breathlessness and faintness; whereas mere dyspeptic palpitation is usually relieved by exercise; the body is usually constipated; there is anorexia, with an especial distaste for animal food, and a predilection for sour articles, as acids, acid fruits, salads, &c.; the catamenia are deficient, and usually replaced by leucorrhœa; or, *what is too often overlooked*, they are profuse, lasting from six to ten days, consisting of blood instead of the normal secretion, and, in fact, constituting a passive hæmorrhage, which is often the cause of the anæmia; the muscular system is very feeble, lassitude and aching pains of the limbs being produced by trifling exertions; the intellectual powers and energies are also greatly impaired; in many patients, there are transitory neuralgic stitches and aches in various parts of the body, and sometimes exquisite sensitiveness of the skin, especially that of the mammæ and abdomen; more or less headache is almost always experienced, generally with vertigo, rushing noises in the ears, and, in severe cases, with intolerance of light and sound, delirium, and even fatal coma, of which I have recently witnessed two instances. Such are the general signs of anæmia, and therefore of anæmic palpitation.

We proceed to the *physical signs*. The impulse of the heart is less remarkable for force, than for an abrupt, bounding character, with throbbing of the arteries—often universal, and a jerking pulse. Hence, this species of palpitation is more audible to the patient than perhaps any other, the sound appearing to rush through his ears, especially when he lies on his side in bed and each arterial throb causes a movement of his pillow. Some are so sensible of the universal arterial throb, that they can count the pulse by the mere sensation, particularly as experienced in the back, when resting against a chair.

When the anæmia is considerable, palpitation occasions a weak, soft bellows-murmur in the aortic orifice,* with the first sound; and a corresponding whiff is heard in the carotids, subclavians, brachials and other considerable arteries, especially when slightly compressed with the edge of the stethoscope, though this is not always essential to the production of the phenomenon. These murmurs in the heart and arteries occur whenever the action of the organ is excited, and in some patients the slightest causes suffice to produce the excitement; as, for instance, a momentary mental emotion, a change of posture from the recumbent to the erect, a constrained position, a meal, flatus in the stomach, &c. I have often found the phenomenon to subsist for a few seconds or minutes only; that is, so long as the exciting cause continued in operation. The patient, if asked whether he is conscious of palpitation, invariably replies in the affirmative; yet the pulse may not be strong—it may even be small and weak; but it will always be "jerking." It is the velocity, therefore, and not the power of the heart's contraction, which, operating on attenuated, aqueous blood, is the active instrument in occasioning the murmur. This subject has already been fully explained (p. 102).

Another phenomenon invariably attends the inorganic murmurs in the heart and arteries, and may even result from a slighter degree of anæmia. I allude to the venous murmur in the jugular veins, particularly the internal. This has already been fully described, (p. 109,) and it has been shown that musical hums and whistles, ascribed by Laennec and Bouillaud to the arteries, are really referable to the veins.

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* It might be expected in the pulmonic orifice also; yet I have not been able to satisfy myself of its existence in this situation. Some cases, however, at present under my care, lead me to think that the point is open for further investigation.

For the diagnosis of anæmic, from valvular murmurs of the heart, the reader is referred to p. 389. The signs there explained, taken in connexion with the general symptoms of anæmia, divest the diagnosis of all difficulty.

Anæmia often complicates dyspeptic, hysteric, and nervous palpitation, and that from organic disease of the heart. In all cases, it is of the utmost importance to detect it; as, unless there be insuperable contra-indications, it always requires a degree of the treatment described below.

The treatment of anæmic palpitation is simple, certain, and satisfactory. The operation of the exciting cause having been suspended, the never-failing remedies, unless there be counteracting complications, are, large doses of any of the stronger preparations of iron, continued for three to six or eight weeks; with aloetic aperients, to maintain a free, but not relaxed state of the body; and a large proportion of animal food, especially mutton and beef, lightly dressed, and taken twice a day at an interval of not less than six hours. A dry, bracing air, a change of air, and out-door exercise short of fatigue, are valuable auxiliaries.

3. Palpitation from too stimulant diet. This I have observed to be a very common affection amongst medical students coming from active avocations and a full diet of animal food, porter and wine, in the country, to sedentary, studious habits in London, without making a change to a lighter diet. I have noticed the same in Oxford and Cambridge men, in young barristers and attornies, and in various others under analogous circumstances. After a few weeks, a state of febrile excitement comes on. The pulse is accelerated and full; the tongue is whitish; the body confined; the skin hot; the face flushed, with throbbing headache and sometimes universal throbbing. The nervous system is very excitable, so that palpitation is induced on slight exertion, mental emotion, &c.; and it is principally this which excites the alarm of medical students.

A single bleeding, a few smart cathartics, and a broth or fish diet for a week or ten days, easily removes this affection. The diet should subsequently be more moderate, with considerable restrictions as to malt liquors, wine, &c.

4. Palpitation from plethora. This occurs principally in those who have a decided plethoric tendency. After living too freely, or relinquishing active habits without a reduction of diet, and sometimes without any very obvious cause, the patient becomes stouter than usual, and complains of palpitation, or undefinable oppressions in the præcordial region, sometimes with slight angina pectoris; these symptoms are increased by exertion, mental excitement, and often by meals; the pulse is small and oppressed; the spirits dejected, sometimes with vague fears or dread of death; constipation and dyspepsia generally attend,—the latter sometimes inflammatory. The symptoms proceed from a gorged state of the heart and whole vascular system.

The diagnosis is formed by the absence of all the usual physical signs of organic disease of the heart. In consequence of the feebleness of the pulse, I have often seen the complaint mistaken for nervous debility, and treated ineffectually with tonics.

The most prompt relief is afforded by bleeding to the extent of eight or ten ounces; which, by removing the vascular tension, restores fulness and strength to the pulse, and often dissipates the angina at once. The depletion may be repeated, if necessary, once or twice, at intervals of two or three weeks. Meanwhile, aperients should be employed, and the diet restricted and regulated according to the dyspeptic symptoms. Dyspepsia is, in fact, a most salutary check on immoderate feeding in those who have a decidedly plethoric tendency, for whom a singularly small quantity of food is generally sufficient.

Such are the principal varieties of inorganic palpitation. So common are they, that, of those who consult in private practice for supposed organic disease of the heart, I have found at least one-half to be exempt from that malady.

Inorganic and organic causes of palpitation not unfrequently coexist, and the discrimination of both is of great importance, as the treatment must undergo corresponding modifications. The practitioner who is well acquainted with the signs of each separately, will experience little difficulty in recognizing the two when combined.

The following cases are added, as exemplifying more graphically than can be done by mere description, a few both of the combined and the separate varieties.

Dilatation with Hypertrophy; over-feeding; simple apoplexy.

A young medical gentleman was subject for four years to dyspeptic symptoms and palpitation of the heart. They commenced about six months after leaving school, where, while growing rapidly, he had been accustomed to very violent exercises and exertions. When he became an apprentice, his appetite was very great. He ate large quantities of animal food, and never took any kind of exercise. His first symptoms were a heavy pulsating pain in the back of the head, extending forwards to the forehead, aggravated by any sudden motion, particularly that of rising up or lying down; giddiness, and disinclination to any exertion, sleep constantly disturbed by frightful dreams, particularly if the head was not much higher than the body; shortness of breath and palpitation on going up stairs or up a hill; a weak fluttering pulse when agitated or startled, accompanied by a sensation of weight and fulness about the heart. These symptoms continued for about three months, when he had a fit of congestive or simple apoplexy, for which he was bled, blistered and purged. He now fell greatly into the habit of rowing, and made long and violent exertions in matches, &c.; but he was always short-winded during the effort, and after it suffered from palpitation. At this time he entirely lost his appetite; his fingers were blue and very generally dead, particularly in the mornings, and his pulse feeble. He was seldom without heart-burn after any meal, and when this was the case he suffered more from palpitations, dreams, &c., and his fingers were more constantly dead. His feet also were nearly always dead, sometimes through the whole night. His bowels were irregular and costive, and skin cold and damp. By being bled occasionally when the affection of the head became worse, by abstaining from all violent exercise, paying attention to diet, correcting dyspepsia by occasional stomachics and antacids, and keeping the bowels open, he completely recovered from the above symptoms within four years from their first appearance, not having employed any systematic medical treatment for the first two. In the course of a year and a half after the commencement, he had few symptoms remaining, and he considered himself quite well, never having any return of palpitation but when he had heart-burn, or had been taking too violent exercise.

In this case, the dyspeptic and congestive symptoms were superadded to hypertrophy with a predominant degree of dilatation, as indicated by auscultation and percussion. Mere nervous affections of the heart, without plethora, never present the marked symptoms of cerebral determination exhibited in the present instance.

Eight years have elapsed since the preceding case was written. From a premature return to active habits, the patient had two recurrences of simple apoplexy. For the last six years, his enlargement of the heart has been cured, and he has enjoyed exemption from all his old symptoms.

Dyspeptic and Nervous Palpitation, with Dilatation and Cerebral Congestion.

A gentleman, æt. 40, has from his youth been very subject to febrile and inflammatory affections, and, though active, he has always been rather short-winded on ascending. He is subject to starting from sleep, in consequence of which he awakes shouting out violently, and always finds himself in a fit of palpitation and great agitation. When merely awakened from sleep by his attendant, he always starts suddenly and in the same state of agitation. For at least ten or fifteen years he has always experienced great confusion in the head on first lying down to sleep: so much so, that he has seldom ventured to retire to bed alone. The same uneasy sensations have occurred on awakening in the morning, and he has always required a quarter of an hour to compose himself before rising. Hence, he has always had a dread both of going to bed and of getting up. He has about eight times had a kind of fit, i. e. an indescribably distressing universal sensation, as if he were dying, invariably accompanied with palpitation and gasping, and terminating in partial unconsciousness, though without any convulsive movements. After an attack of this kind he immediately recovers the perfect command of his faculties. He has never had any paralytic sensations, though he is subject to tingling of the fingers, to a tremulous sensation of the left side and extremities, and to sleeping of the feet. He has occasionally experienced optical illusions, and once lost his sight completely, as if a blanket had fallen before his eyes.

He is subject to a throbbing pain in the posterior part of the head, which is one of his most distressing symptoms, and to acidity and excessive flatulence. A load of undigested and acid food is more apt than any other cause to bring on the fit described, and it is immediately relieved by an emetic, and sometimes even by a large dose of soda. Bowels regular, but evacuations generally unnatural.

Until three years ago, he was in the habit of being cupped on the nape of the neck every three or four months, and experienced great relief from the depletion. Since that time, it has been less necessary, and has only been resorted to occasionally.

The patient gets rid of nearly all the inconvenient symptoms when he pays strict attention to diet and regimen, and relieves the circulation by cupping when the cerebral symptoms indicate it.

This is a case in which the symptoms were so closely connected with stomachic derangement that they were long supposed to result from the latter cause exclusively. The nature of the cerebral symptoms, however, and the evidence of dilatation afforded by auscultation, remove all doubt as to the real nature of the complaint. Since the preceding was written, eight years ago, the patient has enjoyed unusually good health.

Dyspeptic, Hypochondriacal, and Nervous Palpitation.

A gentleman, æt. 22, rather dyspeptic from his youth, became affected with permanent depression of spirits from a mental cause. This was followed by excessive torpor of all the functions both corporeal and mental. Dyspepsia manifested itself in its most aggravated form, while the mind, naturally energetic and powerful, became obtuse and totally incapable of exertion, and the spirits sank into a state of apathetic despondency. With these symptoms he experienced palpitation on the slightest exertion or emotion. It sometimes consisted in merely a few rolling or tumbling movements of the heart, attended with a sensation of fulness and oppression; at other times, the organ fluttered and faltered for several minutes or for a longer period, the pulse being small and feeble, and exhibiting the same unsteadiness; at other times, again, the palpitation amounted to a violent paroxysm accompanied with gasping and orthopnœa. During the intervals of the attacks, he was neither short-winded nor subject to palpitation, and he invariably improved by exercise, which he is capable of taking to a great degree.

He was subject to occasional, temporary, local pains in the head, with stupor, somnolency, and sometimes with visual illusions.

This patient, after suffering for upwards of four years, almost completely recovered, by a removal of the mental depression, by travelling as a pedestrian, by an abstemious dyspeptic diet, by strict attention to maintain regularity of the bowels, by combating fits of dyspepsia immediately on their appearance, (for which evacuation of the stomach by an emetic, followed by abstinence and an aperient, were the most efficient remedies,) and by pills consisting each of a grain of sulphate of iron, one of aloes, and three of comp. cinnamon powder, taken to the extent of one or two whenever the bowels were torpid, a lavement being employed when the pills failed.

In this case, the sounds and action of the heart were natural, except during the attacks of palpitation. The cerebral symptoms were partly nervous, and partly those of a languid circulation through the head, but not of increased determination to it.

The state of the patient's health has continued to improve up to the present date.

Plethoric Dyspepsia, with Palpitation.

A medical gentleman, æt. 35, consulted me in 1838. Very stout and plethoric, became subject to indigestion, with slight dyspnœa, palpitation, and headache. Bleeding invariably relieved these symptoms, and, for a time, made him feel light and comfortable. This condition having continued for ten months, during which period he became much stouter, he was attacked, one morning, on going out, with palpitation, consisting of quick, weak, irregular, and fluttering beats of the heart, with dyspnœa and anxiety. Was not relieved by æther and ammonia, and, as he felt numbness in the right hand, which created apprehensions of apoplexy, he was bled to thirty ounces, but with little immediate relief, as the attack of palpitation continued for two hours. During the ensuing month, he experienced heart-burn, with a "consciousness of having a stomach during digestion;" pimples on the tongue; head-ache, and dyspnœa on ascending. Always felt better after taking aperient and stomachic draughts, even though the body was previously regular. Stated that, being subject to thirst, he had been in the habit of drinking very freely of water and slops; from malt liquors, wine, &c. he had wholly abstained, as they excited him. Had always been a moderate and plain eater. Auscultation proved the heart and lungs to be sound.

I considered the symptoms, in this case, to depend mainly on vascular plethora, though mental emotion had probably contributed. I desired him to abstain, to the utmost, from liquids; to restrict his meals, especially dinner, still more, and to be bled to six or eight ounces occasionally; also to take an alterative aperient pill on alternate nights, and a bitter aperient and antacid draught thrice a day. He speedily improved, and has enjoyed good health up to the present time, that is, upwards of a year.

Plethoric Congestion of the Heart, with Palpitation and slight Dyspepsia.

C——n, Esq. æt. 40, plethoric and red; (13 stones;) has gained two stones in the last two or three years. Eats eight ounces of meat at breakfast and more at dinner, and drinks about half a pint of wine.

Habits sedentary; i. e. writes six or seven hours per day; whereas he formerly had much more exercise.

Complains of a feeling of oppression about the heart, as if it could not contract, or "as if there were a stoppage there." Occasionally has a feeling of something tumbling or bounding in the part (the systole following an intermission). These sensations render him very uncomfortable, yet he can walk up hill and up stairs without dyspnœa or palpitation.

Tongue white, furred; occasionally a little distention of stomach after meals, but no other symptoms of dyspepsia. Bowels, regular daily. *Pulse, small, weak and oppressed.* No head-symptoms except occasional vertigo and increased nervous irritability,—being much more upset than formerly by any business of a public nature.

Auscultation.—First sound very weak, and second weaker than natural, allowance being made for obesity.

Remarks.—Here, the heart and whole vascular system were overdistended, so that the organ could not contract freely and fully. The case shows an incipient degree of functional disturbance of the heart.

He speedily recovered under purgatives, a limited and less stimulant diet, and the use of liq. potassæ as an absorbifacient.

Plethora; dyspepsia; hepatic enlargement; jaundice; intermission; palpitation; "fulness" of the heart; and fainting.

The subjoined letter, graphically descriptive of his own case, is from an eminent practitioner in one of the provincial towns, who laboured under intermission of the pulse, resulting from the anxieties of an active and powerful mind, and the irregularities of diet and hours, inseparable from a very large and successful practice. He has consulted me at intervals since 1834, but on no occasion presented physical signs of organic disease of the heart. He is of a sanguine, excitable temperament, and rather plethoric habit.

"January 18, 1839.

"My dear Sir,—I think you will feel some interest in a brief history of your quondam grateful patient, with supposed affection of the heart, since he last consulted you.

"On returning into the country, I determined, as far as my practice would permit me, to conform to the directions you kindly gave me. I rode less on horseback, I got an active assistant, I sat up less at night, I determined on meeting anxieties with a firmer front, and I married. Still, my dear doctor, the unruly heart jogged on very interruptedly. I began to feed, as happy husbands do, till I made my ten stone five pounds, as it was when you last saw me, into twelve stone nine pounds. I indulged in shooting a little each season, and hunting sometimes—always increasing thereby the palpitation and irregularity, yet, otherwise, with decided benefit to my health.

" I believe that, till within the last month, I was never able to count eighty beats without interruption, and more generally the interruption occurred every five or six beats. There was also considerable pulsation in the jugular veins, and a marked increase in their volume. After exhaustion or privation of sleep, there was the *saw-sound* in the region of the heart, audible to my wife. You may suppose that, during this period, I often reflected on the necessity of putting my house in order, and really calculated on great diminution of ' length of days.' On ascending a hill or a long flight of stairs, I have often felt as though I must there die; and have very often been asked by attendants if I was not very ill. Twice I actually fainted—which you will imagine, with my knowledge of the cause, must have been most distressing. I have on many occasions requested that Mrs. —— would take the opportunity of feeling my pulse whilst sleeping, to ascertain if it was then irregular;—thinking that, when it was working without the influence of the fears of an anxious mind, alive to the dangers of the supplies being not only suspended, but actually cut off, it might beat regularly. No! she found the irregularity still the same!

" For some months back, I have frequently suffered pain in the liver and right shoulder, with indigestion and acidity of stomach; and, a week before this Christmas, I was seized with inflammation of the pyloric end of the stomach, and (I suppose, from the seat of the pain and the uneasiness on anything passing from the stomach) duodenum. Jaundice came on in two days, and I was confined to bed nearly a fortnight.

" A few days after the commencement of my illness, I bethought me of the old enemy, the heart, and felt my pulse:—when, to my great astonishment, it was, though quick, perfectly regular! Many times in that day, I found the same happy regularity. In a fortnight I left home, to avoid the plague of being consulted before I was able to do my duty, and went on a visit to a gentleman who farms his own estate, and there, with some precaution, indulged in my favourite amusement of shooting. On the second day I found I could mount the hills as well as my friend, bag more game, and bear the same exertion with apparently less fatigue:—and all without my old pest, the 'bad heart.'

"It is now a month since I was taken ill, and I believe no day has passed, up to the present, without my examining (be assured more than once) my old enemy, to see if he was vanquished, nor have I once detected any irregularity in the heart's action. The miserable *fulness* about the heart has left me. I can walk up stairs without fatigue, and, returning from —— to night, I ran a mile to ascertain whether that would produce the interruption; but all, thank God, is once more quite right.

"I took small doses of calomel, under Dr. ——'s direction, during the acute part of the attack, and also mild saline aperients; but it is worth while to remark that, during my illness, I have lived most abstemiously. I have avoided my besetting sin, coffee-drinking; also wine and beer; have reduced my animal diet very considerably, have avoided strong tea, taken daily some walking exercise, and am reduced fourteen pounds in weight.

"My grateful feeling for your former kindness has induced me to trouble you with this narrative, hoping you may find some interest in it. At your leisure, I should be very happy to hear from you," &c.

I heard from the writer three months after the date of the preceding letter, and he continued "enjoying a perfectly regular pulsation and excellent health and spirits." The *saw-sound* had ceased.

Remarks.—This is a striking instance of the extent to which functional derangement of the heart may proceed in a plethoric individual, over-excited by intellectual exertions, and by too full stimulating a diet, taken at irregular hours.

I have repeatedly heard the arterial murmur, (the saw-sound of my correspondent,) during states of excitement, in individuals of the sanguine temperament, even though not anæmic; for their blood is naturally thinner than that of melancholic temperaments. I presume, therefore, that this was the cause of the phenomenon. It did not exist when I examined him.

The good effects of the reducing treatment employed for the gastro-duodenitis and icterus, strikingly display the advantages of effectually disgorging the whole vascular and hepatic system in such cases.

The following case is analogous to the preceding.

Plethora; bilious engorgement; intermission of the pulse; occasional fainting; great oppression and debility.

A lady, æt. about 40, of large, full habit, consulted me for supposed disease of the heart in 1838. The pulse was feeble, and presented three or four intermissions per minute. She felt great oppression in the præcordial region, with faintness,—especially on lying down. She was totally unable to ascend a flight of stairs, as the effort produced overwhelming faintness, with fluttering palpitation, &c. I discovered no physical signs of organic disease of the heart; but, on examination, I found the liver enlarged, and the alvine evacuations bilious. During the whole previous year, she had experienced great constipation.

Active mercurial purgatives, employed almost without intermission for two months, brought off an incredible quantity of dark green and deep orange bile. If the medicine was at any time suspended for a couple of days, for the purpose of examining the evacuations uninfluenced by calomel, she felt worse—nor were the motions exempt from bile. Her diet was principally veal and chicken broth, and farinaceous articles. At the expiration of two months, the evacuations were healthy; all intermission, palpitation, and faintness had ceased; and, though thinner and paler, she felt light and active, and ascended a long staircase with perfect ease. I saw her three months afterwards in the enjoyment of perfect health—"much better than she had been for years."

Remarks.—The symptoms, in the present case, were mainly dependent on the poisonous effects of bile; but the reduction of plethora probably contributed to her restoration. Bilious accumulations in plethoric free livers, with constipation, are very apt to be overlooked; and supposed debility often scares practitioners from adequate purging, even if they are not deterred by imaginary disease of the heart. The advantage of good diagnosis of cardiac disease in such cases, is obvious: without it, the practitioner is timid and undecided.

The following case is another aspect of an analogous affection.

Derangement of the stomach, bowels, and liver: paroxysm of palpitation, with orthopnœa.

A distinguished surgeon of the metropolis called on me late at night, in 1834. As I happened to be out, he returned home, passed a sleepless night in a state of orthopnœa, with great præcordial oppression, and sent for me very early on the following morning. I found the pulse and action of the heart to be small, weak, irregular, intermittent, and unequal, in as great a degree as I have ever seen them in the worst cases of disease of the valves. He was in alarm lest rupture of a valve or great vessel had taken place, as the symptoms had supervened rather suddenly.

On careful examination, I found the sounds and impulse of the heart perfectly natural, except the irregularity. The complaint was traced to unusual professional exertions, with too full and indiscriminate a diet, which had deranged the alimentary mucous membrane and the liver. An active mercurial cathartic afforded almost immediate relief; and alterative aperients, a restricted diet, and light antacid bitters, restored him to perfect health in the course of ten days. He has not subsequently had any recurrence of the same affection.

CHAPTER III.

SYNCOPE.

Opposed to the state of over-excitement of the nerves of the heart, which we have been considering in the last chapters, is that of deficient excitement, the extreme degree of which constitutes *syncope.* Numerous agents have the effect of reducing, and even completely suspending, the contractile power of the heart. Such, for instance, are the depressing passions, feelings of disgust, certain scents, pain, violent shocks of the nervous system from accidents, sudden loss of blood, suddenly raising the patient to the erect position in cases of great anæmia; congestion of the heart from obstacles to the circulation; stupifying poisons, as hydrocyanic acid, digitalis; certain miasms, as the plague, Indian cholera, pestilential fever; any agents, in short, which can, directly or indirectly, suspend for the moment the excitability of the heart.

The phenomena of syncope are too well known to require description: it may be said summarily that they are those of sudden death, except that, in most cases, though not in all, the patient can be restored to life. The ordinary duration of syncope is from a few seconds to a few minutes; but in certain rare cases it lasts for hours and even days, sometimes imitating death so perfectly as to lead to the horror-striking accident of living inhumation. In such cases, however, the action of the heart is not wholly suspended, though exceedingly feeble. I imagine that the second sound would be heard with the stethoscope, though possibly the first might not. In ordinary cases of syncope, the unconsciousness is seldom complete, and, though the pulse be imperceptible, feeble sounds of the heart's action may in general be distinctly heard. The latter is sometimes the case in individuals, who, after immersion in water, or other causes of asphyxia, exhibit no apparent signs of life. Under these circumstances, therefore, auscultation should invariably be employed; for, so long as the sounds are heard, the patient is perfectly within the possibility of recovery.

Syncope, though free from danger when purely nervous, is a formidable accident when accompanying organic disease of the heart, as it is apt to terminate in sudden death, being, in fact, less the cause than the symptom of a fatal suspension of the circulation. This catastrophe is more liable to occur when angina pectoris is superadded to organic disease; in consequence, apparently, of the lesion being double, the motive principle as well as the muscular apparatus of the heart being inadequate to the discharge of its function. Sudden death is also apt to occur from the syncope of anæmia, especially when the patient is suddenly raised erect.

Treatment.—The ordinary excitants, which suffice for so slight an affection as purely nervous syncope, are, the horizontal position with the head low, fresh air, the sudden aspersion of cold water, startling the patient by a sudden noise or blow, ammonia and other pungent errhines. When syncope is symptomatic of another disease, it requires that, in addition to its ordinary treatment, remedies should be employed, adapted to the nature of the primary affection. The principles according to which this must be done in reference to disease of the heart and angina pectoris, have been explained in the preceding pages. In extreme cases of anæmia, the patient should be kept constantly in the horizontal position, till the tendency to fainting has ceased. This remark applies to those more especially, who have sustained a great loss of blood. The treatment of anæmia has been summarily described in the preceding chapter, p. 510.

PART V.

MISCELLANEOUS AFFECTIONS.

THESE affections consist of a few which are not reducible to any of the preceding heads.

CHAPTER I.

POLYPUS OF THE HEART.

THE concretions of blood commonly called polypi of the heart and great vessels have given rise to much discussion, and various doctrines respecting them have successively superseded each other in the schools. According to some, they are merely coagula of blood formed after death: according to others, they are organized substances, formed before death, and analogous to nasal and uterine polypi: others, again, believing that both kinds existed, denominated the former *false* and the latter *true*.

It was a very general opinion during the last century, that polypi produced all the symptoms which are known to result from organic disease of the heart: while some, on the contrary, doubted whether they produced any symptoms whatever. The researches of Corvisart, Testa, Burns, Kreysig, Laennec and succeeding pathologists have decided the question, and have fully established the fact, that there are some polypi formed during and after dissolution, and others formed for a longer or shorter period anterior to it, presenting various degrees of organization, and the cause of certain well-marked symptoms during life. These facts have more recently been corroborated by Bouillaud, who gives the results of sixty-five cases seen by himself, M. Legrouse, or others (Traité, ii. p. 592, 1835).

That polypi should form before death, might be anticipated *a priori* from the fact that, in the arteries and veins, blood can coagulate during life, and, becoming organized and adherent to the walls, obliterate the canal of the vessel. Instances of this have, of late years, been accumulated in abundance by Hodgson, Burns, Kreysig, Bertin and Bouillaud, Laennec, Velpeau, Cruveilhier, Mr. Arnott, Dr. Robert Lee, and the writer: in short, there is scarcely a single considerable vessel, especially a vein, in which the concretions in question have not been found. In veins, they are a well-known cause of partial dropsies;* as the white swelled-leg or *phlegmatia dolens*, from obliteration of the femoral vein.

It has long been known that polypi are of more frequent occurrence in the right, than in the left, side of the heart: M. Bouillaud adds, on the faith of the cases which he adduces, that they are also more frequent in the auricles than in the ventricles. The principal cause of this evidently is, that the blood is more easily retarded and rendered stagnant in the right cavities, and that it is in them especially that it accumulates during the last period of life and after death. M. Bouillaud is of opinion that other causes also may explain the circumstance: such as, the frequency of phlebitis, which is sometimes propagated even into the right cavities; perhaps, a more marked tendency to coagulation in the venous, than in the arterial blood, &c. (Traité, ii. 608).

Anatomical Characters of Polypi.—These will be rendered more simple by considering the polypi as, 1. *unorganized*, 2. *slightly organized*, and 3. *more completely organized.*

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* Vid. M. Bouillaud Archiv. Gen. de Med. tom. ii. et v.

1. *Unorganised Polypi.*—Polypi formed after death, or during the last moments of life, are concretions of fibrine, which, if very recent, merely overspread portions, but seldom the whole, of the clots of blood in the heart and great vessels with a thin, translucent layer resembling inflammatory buff: but, if rather older, they constitute larger and thicker masses, often entirely independent of the red clots of blood. In dropsical subjects, or when the blood is very serous, the fibrine appears as it were infiltrated, and is soft, trembling, and semi-transparent like jelly. Polypi of the above kinds are far more common on the right side of the heart than the left; they do not adhere to the walls; they are of a uniform semi-transparent yellowish or whitish colour; and they do not present any trace of internal organization and structural arrangement: by these characters they may be distinguished from polypi formed some time previous to death.

2. *Slightly organized Polypi.*—It may be premised, as a fact ascertained by observation, that fibrine, separated from the blood and become concrete in a living organ, (whether the heart, the blood-vessels, or serous, cellular, or other tissues into which it had been extravasated,) retains its vitality and is susceptible of organization in an equal degree with inflammatory lymph.

Polypi formed some time before death, in which this organization has commenced, are of a much firmer consistence; more opake, and less charged with serum; their fibrous texture is more distinct; they are often arranged in concentric layers; their colour, instead of being uniformly whitish or yellowish, has in parts a pale flesh tint sometimes slightly violet, from incipient vascularity; they are found more frequently on the left side of the heart than recent polypi are; and they adhere more or less firmly to the walls of the heart, from which it is scarcely possible to draw them away in a single piece, as the extremities remain attached under the columnæ carneæ. The medium of adhesion is often a filamentous tissue, the rupture of which leaves a roughness both on the lining membrane of the heart and on the surface of the polypus. The surface also presents spots of blood penetrating more or less deeply and sometimes ramifying inwards, as if to form vessels for the purpose of organizing the mass. Some of these polypi contain pus in the centre, sometimes pure, at others, curdy or sanious—precisely what we so commonly see within coagula formed by phlebitis (see the writer's Morbid Anat. Figs. 204 and 240). The *globular vegetations* of M. Laennec (De l'Auscult. ii. p. 530) are, in my opinion, nothing more than varieties of these suppurating polypous concretions. They present themselves under the form of irregularly spherical or ovoid balls or cysts, the size of which varies from that of a pea to that of a pigeon's egg. The cysts are smooth externally; and their walls, which scarcely exceed half a line in thickness, are composed of an organized substance somewhat firmer than the white of a hard-boiled egg, and resembling in opacity the oldest polypous concretions. The internal surface of the cyst is less smooth than its exterior, and appears formed of a softer substance, which sometimes gradually degenerates, in the direction from without to within, into a matter similar to the contents of the cyst. These contents, in the cysts which there is reason to believe the most recent, are bloody; in the older they are like lees of wine, and in the oldest they are puriform. The cyst adheres by a pedicle, which, according to M. Laennec, is of more recent formation than the cyst itself, being more translucent, and in a less advanced state of organization. The pedicle is interlaced amongst the columnæ carneæ, and united more or less firmly with the internal membrane. The most common situation for these bodies, and where I have frequently found them, is about the apex of the ventricles. I am not aware that they are ever found in the great vessels: I have never seen them there.*

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* Respecting the source of the pus within polypi, M. Bouillaud gives the following opinion. "Various authors, and M. Legroux in particular, regard this pus as a product of inflammation of the concretion which contains it. 'An inflammatory movement,' says M. Legroux, shows itself in the concretion . . . . it softens in the centre, becomes granulated, passes to the sanious, then to the purulent state: subsequently, the pus is absorbed and there only remain the exterior layers of the concretion, which have resisted the softening, and which form the walls of the abscess, or the cyst.' As for myself, pursues M. Bouillaud, I think that such is not the ordinary origin of the pus which is found in concretions: this pus appears to me to have been either secreted in the cavity of the heart, or to have been transported thither by absorption, and then to have occasioned the formation of a coagulum which has entirely enveloped it. At the period when pus in the centre of a concretion is most frequently found, the concretion presents scarcely the rudiments of organization, and one can hardly conceive how, in this stage, it could undergo an inflammation characterized by

3. *More completely organised Polypi*.—There are other polypi which appear to be of still older formation, and which may probably be dated as far back as several months prior to the death of the patient. They are completely opake like paste or cheese, exactly resemble the oldest fibrinous layers of false aneurisms, and adhere so firmly to the walls of the heart that they cannot be detached without scraping with the scalpel, and sometimes without removing the internal membrane.

Causes and formation of Polypous Concretions.—Two opinions have been entertained respecting the formation of polypi: 1. Some have attributed them to retardation of the blood, an entirely physical cause. 2. Others have ascribed them to inflammation; that is, in other words, to causes acting chemically or vitally on the blood. Modern experience shows that both of these opinions are correct.

1. When polypi result from mechanical retardation and consequent stagnation of the blood, we find them to occur under circumstances the most favourable to that stagnation: namely, during the last hours or days of waning life in all diseases—especially chronic diseases which have occasioned cachexy, emaciation, extreme debility, or which have been accompanied by any considerable obstacle to the general circulation; for instance, dilatation with attenuation, softening, or great valvular disease of the heart. Under these circumstances, so retarded is the circulation that blood will scarcely flow from the veins, opened by the lancet, and it sometimes actually coagulates in them. I have

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purulent secretion. I do not pretend to say, however, that, when once well organized, sanguineous concretions may not inflame and suppurate. Nevertheless, this is not, if I do not mistake, a very common occurrence" (Traité, ii. p. 610).

To myself, the opinion which ascribes the pus to inflammation appears the more probable, as being more in accordance with the suppuration of coagula which we constantly see in phlebitis, and also because I think that pus in the circulation would be mixed *equally* with the blood, and not collected in particular points, as supposed by M. Bouillaud's theory. I do not even believe that what are called *purulent depositions* in organs really consist of particles of pus deposited by the blood; but that pus poisons the blood, and occasions its coagulation and suppuration in the spots affected. In this point of view, I do not deny that particles of pus may be the nuclei of coagula within the heart; for I have seen such coagula, some suppurating and others not, floating loose in almost every considerable venous trunk through the system of the same patient (See the writer's Morbid Anat. Fig. and case 110).

taken notes of a number of cases of phthisis, in which this coagulation took place in the femoral veins, and caused œdema of one or both extremities. That stagnation alone suffices to cause coagulation, is a fact too familiarly known to require demonstration. We see it, out of the body, in blood drawn by the lancet: we see it exemplified within the body, by the fibrinous concretions that fill up false aneurisms; the operation for this disease, moreover, has for its basis the coagulation in question.

The adhesion of polypi from stagnation appears to be occasioned by the irritating action of the body itself on the walls of the heart; whence there results an exudation of lymph on the latter, which forms the agglutinating medium. I once saw this process strikingly exemplified in the veins. Loose coagula were found in most parts of the venous system; but, in the vena portæ, they were adherent wherever larger trunks, subdividing into others too small to admit them, had arrested their progress.

2. The knowledge of the chemical or vital causes of coagulation of the blood is one of the improvements of modern medical science. It is well ascertained, that when the walls of a vein or artery are inflamed at any particular spot, the first effect of that inflammation is, to cause coagulation of the blood within the vessel at the inflamed part; and here it is to be presumed that the inflammation exercises some *vital* influence over the constitution of the blood within its reach, disposing it to coagulate. If this can take place within the blood-vessels, it is consistent to suppose that it may occur equally within the heart, when the interior of the organ is inflamed; accordingly, the cases of fatal acute endocarditis which M. Bouillaud has met with, and in which he has found polypi evidently formed some time before death, afford strong evidence that such is actually the case. This writer, moreover, is of opinion that a *general* inflammatory condition of the blood, dependent on "*any* pure inflammation in which there is violent fever, and where the blood drawn from a vein presents a good, firm, elastic, resistant buffy crust, constitutes a real predisposition to certain fibrinous concretions of the heart, which have then a great resemblance to the inflammatory buff." Thus, on

referring to the cases in his work, "it will be seen," says he, "that in most of the instances in which the fibrinous concretions did not proceed from a mere embarrassment of the circulation, they accompanied, either an idiopathic inflammation of the heart, or an inflammation of another organ, which reacted smartly on the heart, as well as on the whole circulatory system and mass of blood" (Traité, ii. p. 612). This doctrine is far from improbable; yet it will, I think, require for its establishment a greater number of cases than are recorded in the work of M. Bouillaud; for we must not come too hastily to the conclusion in question, when we consider how frequent are acute inflammations, and how comparatively rare is their termination in polypus.

Another well-known cause of coagulation of the blood by a vital or chemical influence, is, pus introduced into the circulation, whence proceed visceral abscesses, typhoid symptoms, &c. Experiment has shown that the same effect is produced by the introduction of various foreign substances into the blood, as mercury, acids, &c.

Signs and diagnosis of Polypi of the Heart.—I stated above that symptoms which are now known to depend on organic disease of the heart, were formerly attributed solely to polypi; this error arose from physicians not being sufficiently acquainted with morbid anatomy to recognise organic disease of the heart in those individuals in whom, after the existence of the symptoms in question, they discovered polypi.

The effect of polypi is, to cause a greater or less obstacle to the circulation according to their size and situation. I have generally found those filling up an auricle produce this effect in a greater degree than any others, probably because the auricle, from having less contractile power to expel the stagnating blood, gets more completely charged with the concretions, and partly also because auricular polypi usually send off prolongations or projections into the orifices, which not only impede the action of the valves, but also choke up the passage. When polypi form suddenly a short time previous to death, as within a week or ten days, they exceedingly aggravate all the symptoms of an impeded circulation; and this they do, both in diseases in general, and more especially in diseases of the heart. When, in the latter, they nearly obliterate the cavities or orifices of the heart, they prove rapidly fatal.

Physical Signs.—M. Laennec thinks that polypi of considerable magnitude may be recognised by the following physical signs. "When, in a patient who, till then, had presented regular pulsations of the heart, these suddenly become so anomalous, confused, and obscure, that they can no longer be analysed, we may suspect the formation of a polypous concretion" (De l'Auscult. tom. ii. p. 597). The obscurity of the sounds proceeds from the play of the valves being impeded. I have not found any murmur attend the sounds, but others have, in a few rare instances; and I conceive it possible, if the polypus should happen to entangle a valve while the current through the auricles and ventricles remains tolerably free. Before, however, the murmur can be assumed as a sign of polypus in a given case, it must be proved 1. that it did not previously exist, and 2. that it is not a result of valvular tumefaction from acute endocarditis. Whether the murmur be musical or not, is unimportant.

But though the irregularity described by Laennec be a sign of polypus in cases where the action of the heart was previously regular, it has not the same value in cases where this previous regularity did not exist, and such cases form a large proportion of the instances in which polypus occurs. If, however, even in the latter cases, the irregularity be suddenly aggravated—become unusually "anomalous, confused and obscure;" and if, together with this aggravation, the *general* signs be taken into consideration, the diagnosis may, I presume to think, be almost always formed with accuracy.

General Signs.—The general signs, according to my observation, (for they were wholly unnoticed by Laennec, and scarcely glanced at by Bertin and Bouillaud,) are, a sudden and excessive aggravation of the dyspnœa, without any other obvious adequate cause; the pulse is small, weak, irregular, intermittent, and unequal; the patient is in an agony from an intolerable sense of suffocation; he cannot lie for a moment, and he continues tossing about in the most restless and distressed condition until his sufferings are terminated by death. During this state the surface and extremities are cold, the complexion livid, and, in most cases, there is nausea, and vomiting of all ingesta. To this category, M. Bouillaud has added stupor and slight convulsive movements, which supervened in one of his cases. It has been explained at p. 214, that these same signs occur when there is an *extreme* obstruction to the circulation through the heart, whatever be its cause.

Polypi, formed a considerable period previous to death, are not so easily detected, their deposition being more gradual. Still, if symptoms of the above kind, both physical and general, come on more rapidly than can be accounted for by the ordinary progress of the disease, or if they are such as the disease could not be supposed capable of producing, there is strong reason to suspect a polypus.

The small *globular* polypi often exist without producing any obstacle to the circulation, or any irregularity of the action of the heart. In general, however, they are found in those who have been in a moribund state for many days and sometimes many weeks before death.

Treatment of Polypus.—The treatment is mainly preventive; since, when the concretion is once formed, the case is almost hopeless. One of the greatest dangers of excessive blood-letting or otherwise reducing the system, and of the indiscreet exhibition of nauseants and digitalis, in advanced stages of organic disease of the heart, arises from the risk of the formation of polypi in consequence of languor of the circulation. I am satisfied that this circumstance is not in general sufficiently attended to, even up to the present time.*

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* I cannot but protest against the indiscriminate, I had almost said random, manner in which M. Bouillaud advocates blood-letting for the prevention of polypus. Without specifying any of the circumstances which should guide its employment, he says in round terms, "To prevent the formation of concretions of blood *in diseases of the heart*, whose property it is to impede the current of the blood, it is useful to employ blood-letting from time to time, and to dilute the blood in a manner by aqueous beverages." Now, I have seen enough of blood-letting in this country, (where practitioners have never been very shy of its employment as an empirical mode of relieving severe attacks of dyspnœa from whatever cause,) to know that, in dilatation of the heart, in softening, and in advanced cases of valvular disease, blood-letting will not only fail to prevent polypi, but will actually induce them, as stated in the text, and moreover will favour the supervention of dropsy, exhaust the vital powers, and hurry the case to its fatal termination. Moderate blood-letting may, indeed, be admissible

The best mode, according to my observation, of obviating polypus in advanced cases of organic disease of the heart, is, to keep the patient in a state of the *utmost possible* tranquillity, and in the easiest attainable position, so that the circulation may not become embarrassed from being hurried; to avoid not only nauseants and digitalis, but any other unpalatable remedies which disgust or derange the stomach; to avoid, for the same reason, any but the most simple and digestible articles of diet, and not to introduce much into the stomach at once; for the action of the heart invariably becomes disturbed whenever the stomach is considerably distended either by food or, what is almost as bad, by flatulence, the effect of both being to prevent the descent of the diaphraghm, in addition to their influence through the medium of the nervous system. Though the administration of aqueous drinks, with the view of diminishing the coagulability of the blood by dilution, is plausible in theory, I have generally found it inadmissible in practice beyond a moderate extent, in consequence of the intolerable flatulence which it is apt to generate. Nor must it be forgotten that nature often contradicts the very principle itself; for, while the practitioner is diluting the blood, she is often doing her utmost to get rid of that dilution in the form of dropsy; and that her measures are often the wisest, no one will deny who has observed the great relief to the vascular and respiratory system, which frequently follows a considerable serous infiltration. There can be no doubt, indeed, that dropsy, under these circumstances, is a curative effort of nature.

Such are the negative means of obviating polypus; but there are others of a positive nature, to which the practitioner may

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in the early stages of hypertrophy, even when complicated with valvular disease, but these are not the cases in which polypus is apt to occur.

Again, "blood-letting," adds M. Bouillaud, "is likewise the best means that can be employed against concretions of the heart *already formed*. It has succeeded beyond my hopes in a female admitted under my care (Ward St. Magdalen, No. 3), the 7th of this month (May 1835). A prey to the most imminent suffocation, and offering, moreover, the physical signs of polypus, such as I have explained them above, she has been bled three times, and is at this moment (May 25th) in a satisfactory state" (Traité, ii. p. 618). Thus, without offering more authority than a single case, and that still in the wards, he offers an unqualified recommendation of blood-letting! Surely this is hasty generalization.

resort with advantage. The general surface and especially the extremities should he kept comfortably warm, so as, by diffusing the circulation, to prevent congestion in the heart and great vessels. At the same time, cool, fresh air may be admitted to the head, as this often wonderfully alleviates the craving for breath and consequent restlessness of the patient. On the same principle, the use of the fan is most agreeable. Of medicines, I have found those containing sp. æth. sulph. comp. and ammoniæ sesquicarbonas the most generally useful—probably because, as diffusible stimulants, they distribute and equalise the circulation. In circumstances of great debility, the addition of more permanent stimulants, wine or brandy, becomes indispensable. When paroxysms of congestion of the heart come on, indicated by unusually confused, irregular action of the organ, with an exceedingly small, weak, irregular pulse and suffocative dyspnœa, no remedy affords so much relief as a foot-bath up to the knees, at as a high temperature as the patient can bear it. If he cannot move, the same may be accomplished with much less fatigue by wringing a small blanket out of hot water and surrounding his legs with it up to the knees, discomfort being prevented by enveloping the whole in india-rubber cloth. This may be repeated two, three, or even four times a day, if urgently required, the legs, in the intervals, being kept warm with flannel.

M. Legroux has suggested the use of the preparations of soda and potass, as having a solvent effect on the blood. They certainly render the blood florid out of the body, and the experience of Dr. Stephens in yellow, and other typhoid fevers, and of many in this country in malignant cholera, render it highly probable that they have some corresponding, or at least salutary, effect on the blood within the vessels. Dr. Stephens gives a combination of the carbonates of potass and soda and the chlorate of potass. Further observation is necessary to prove whether these remedies are calculated to obviate polypus.

Such is the treatment when the case is not connected with inflammation: when it is, the inflammation itself must be treated; and if the measures which have been already recommended for pericarditis and endocarditis be adopted, I believe, according to my own observation, that polypus will be of very rare occurrence.

Can polypi, once formed, be dissolved? M. Bouillaud answers this question in the affirmative. "It appears to me indubitable," says he, "that concretions of recent formation and small volume are susceptible of this mode of termination" (Traité, ii. p. 618): but it may fairly be asked whether it is possible indubitably to ascertain the existence of a concretion of "small volume." Organized and adherent polypi are, of course, unsusceptible of solution.

CHAPTER II.

DISPLACEMENTS OF THE HEART.

THE heart being sustained in its place principally by the equal pressure of the lungs on all sides, may be displaced when that pressure is rendered unequal. I have seen this occur from pneumothorax, by which the organ was forced completely to the right of the sternum; by the same affection with hydrothorax producing a similar effect; by hydrothorax alone (case of Rowe and Mitchell); by inflammatory pleuritic effusion, both acute and chronic; by aneurism of the ascending aorta, displacing it to the left (case of Hill); by extreme enlargement of the liver; and by enormous fungus hæmatodes of the right lung. It may also be displaced by emphysema of the lungs, being pushed to the opposite side when a single lung is emphysematous, and into the epigastrium when both are affected: also, by tumors in the anterior mediastinum, and by aneurisms of the arch of the aorta. The two latter causes generally force it downwards. When the heart is enlarged, it is displaced by its own gravitation to a lower situation than natural.

I at present attend a young lady, Miss M., in whom the heart was forced entirely over to the right of the sternum by pleuritic effusion in the left pleura. The aorta was felt to pulsate between the second and third right ribs, an inch from the sternum, and here a murmur was heard with the first sound, which has ceased since the heart has been restored to its natural situation by the absorption of the fluid. Is it therefore possible that a twist given to the aorta, or pressure of the vessel against the ribs, may be the cause of a murmur under such circumstances?

I have at present two cases of still greater displacement of the heart to the right, in consequence of universal consolidation and contraction of the right lung, and hypertrophy of the left. The ascending aorta beats between the second and third right ribs, two and a half inches from the sternum, in one case, (a man æt. 40,) and one and a half to two, in the other (Phœbe James, see p. 115). There is a murmur with the second sound, from aortic regurgitation, in the former case. It remains to be seen whether regurgitation proceeds from a twist in the aorta, disabling the valves, or from disease of the valves themselves. The pulsation of the aorta so far on the right, might be, and actually was, mistaken by non-auscultators for an aneurism.

When the heart is displaced to the right just so far as to be impacted between the sternum and the spine, I have found its impulse to be considerably increased, so as to convey the idea of hypertrophy. This occurred in the case of Miss M. above described, and, until I pointed out the circumstance, the disease was mistaken for hypertrophy, the pleuritic effusion being overlooked. The phenomenon results from the spine presenting an unyielding fulcrum behind. I have already shown that the same occurs in adhesion of the pericardium (p. 194), and in cases of solid tumors, as aneurisms, immediately behind the heart (p. 449). Dr. Stokes has also observed it in cases of tubercular consolidation of the lung behind the heart.

Symptoms.—Slight displacements occasion little inconvenience: when considerable, they may create serious functional derangement, especially palpitation.

Diagnosis.—Displacements are easily detected by auscultation and percussion. The situation of the apex may generally be discovered by its impulse and the usual dulness on percussion: that of the semilunar valves may be detected by tracing the second sound to the point where it is loudest. When the ascending aorta is displaced from beneath the sternum, as in the above three cases, its impulse may be felt between the second and third ribs.

CHAPTER III.

HYDROPERICARDIUM.

Serous effusion in the pericardium is common as an attendant of general dropsy, but very rare as an idiopathic disease. I doubt, indeed, whether there is such a disease as *acute* hydropericardium independent of inflammation. I never met with a case, nor have I been able to find unequivocal instances recorded by authors, the bulk of those reported as such evidently being nothing more than serous effusion from pericarditis. When the fluid does not exceed three or four ounces in cases in which the hydropic diathesis prevails, it may be merely an exudation which has taken place during the last period of life; and when it does not exceed one or two ounces in ordinary cases, it may be ascribed to the same cause.

In general dropsy, the pericardium usually contains less fluid, in proportion, than other serous cavities. I have never seen the quantity amount to a pint: Corvisart states that he has once seen it amount to eight; but I suspect that this was a case of chronic pericarditis. The fluid is sometimes colourless, but usually it is yellowish or brownish, though transparent and free from albuminous flakes; occasionally, though very rarely, it is bloody.

Signs and Diagnosis.—The signs of hydropericardium given by authors are obscure. The weight in the region of the heart, the sensation of the organ floating, experienced by the patient, undulations as of fluid, felt and even seen in the intervals between the third, fourth, and fifth ribs, irregular action of the heart, a small, frequent and intermittent pulse, orthopnœa, palpitation, and syncope, are signs common to other complaints, and therefore unworthy of confidence, except as corroborating others more characteristic.

Laennec expresses himself unable to say what signs auscultation will supply, but thinks that effusions less than a pint will not afford any; and that we shall probably never be able to detect hydropericardium which is not even much more considerable. After much attention to this subject, I think it is in general possible to detect from eight or ten ounces upwards* by the following signs.

Dulness on percussion is preternaturally extensive, and I have known it mount under the sternum, in a conical form, as high as the second rib; the motions of the heart as perceptible beyond the ordinary limits; the impulse is of an undulatory nature, some beats being stronger than others, and the point at which they are most sensible, varying every moment; the impulse does not accurately coincide with the sound of the ventricular contraction, as the heart has to remove the interposed fluid before it can impinge against the thoracic walls; the first sound is dull and remote, in consequence of the intervention of the fluid; finally, the sensation communicated to the hand and the stethoscope is that of an impulse transmitted through a fluid, and not of an organ striking the ribs *immediately*. When the quantity of fluid is very great and the action of the heart feeble, the impulse, I have found in several instances, may be totally imperceptible: in which case the signs are, the unusually extensive, conical dulness—greater than can be accounted for by hypertrophy, and the dulness and remoteness of the first sound opposite to the apex of the heart.

Hydropericardium from general dropsy requires the same treatment as the dropsy. For reputed idiopathic hydropericardium, (which, as above stated, is apparently nothing more than chronic pericarditis,) tapping has been suggested by Senac, countenanced by Laennec, and practised, but unsuccessfully, by Desault and others. Laennec thinks that the least dangerous mode of operation would be, that of trepanning the sternum above the xiphoid cartilage, as, thus, the pleura would not be opened, and the diagnosis might be verified by inspection before the pericardium was punctured. To myself the operation appears inadmissible; for, independent of its danger, unless adhesion were effected by exciting pericarditis, the fluid would probably be regenerated, as in dydrocele and ascites.

* In the cases of Bryant and Snowden a much less quantity was detected, but I would not venture to say that so little could always be recognised.

CHAPTER IV.

PNEUMOPERICARDIUM.

LAENNEC assigns this name to effusions of air within the pericardium, which are very frequently found on dissection. In subjects that have been kept for some time, the effusion is to be ascribed to decomposition; but in many others, judging from the absence of all signs of putrefaction, it is anterior to death. Most frequently, it is conjoined with a liquid effusion, and the two may take place simultaneously in the last moments of life in any disease. Laennec states that he has sometimes detected it by an unusually clear resonance at the base of the sternum which had supervened within a few days, or by a sound of fluctuation attending the beats of the heart and strong inspirations. Though he has not had an opportunity of verifying the fact, he is convinced that, in almost all cases in which the beats of the heart can be heard at a certain distance from the chest, this phenomenon is due to the temporary effusion of a gas, which is in general promptly absorbed, and the presence of which in the pericardium creates no serious inconvenience (De l'Auscult. tom. ii. p. 672 and 455). This is very questionable. I have never, indeed, been able to verify any of the above remarks. Wind and fluid in the stomach might deceive a less cautious observer than Laennec.

Air is sometimes found on dissection in the cavities of the heart. Dr. Forbes of Chichester favoured me, in 1830, with the following communication: "I yesterday examined a boy who had died suddenly, after being affected for years with all the symptoms of extreme dilatation of the heart. I found the organ very large from dilation of both ventricles, and both were distended with air—in all eight or ten ounces. There was no particular putridity, the boy having been dead only thirty-six hours." A similar case is recorded in Simmons's London Medical Journal, part iii. for 1785. As air in the ventricles is incompatible with the maintenance of life, it must, in these cases, have been generated, or conveyed there, after death; but if putrefaction be its cause, it is remarkable that the phenomenon is so rare.

PART VI.

CASES.

The following cases, together with those scattered throughout the work, though few in number, present well-characterised exemplifications of nearly all the ordinary, as well as the more rare diseases of the heart: also of the general histories and signs given in the antecedent parts of the work. I have, for the sake of brevity, omitted the physical signs of pulmonary affections, but have in general adverted to the affections in the diagnosis, and succinctly described them in the post-mortem examinations. I have likewise omitted details of treatment; for, though preeminently important to the observer, they afford comparatively little instruction to the mere reader, by whom, in consequence, they are seldom perused.

Having found it impossible perfectly to classify the cases under the heads of hypertrophy, dilatation, valvular disease, &c., in consequence of these affections being in general complicated with each other, I have merely thrown the several classes rudely together, as far as practicable, and have given an alphabetical index to the names of the patients, which will afford every facility of reference.* A few of the less complete cases of the former

* It may be stated, in reference to the cases taken in St. George's Hospital anterior to 1831, that minutes of the physical signs of disease of the heart were written by other gentlemen in the hospital, as well as myself: more especially by Mr. Johnson, then house-surgeon to the institution;—a gentleman no less remarkable for an intimate knowledge of auscultation, than for general talent combined with a sound

editions I have struck out of the present, and substituted others of an interesting nature, and illustrating particular points, for the most part new.

Great Hypertrophy with Dilatation; Hydropericardium; Emphysema; Peripneumony.

Robert Bryant, æt. forty-two, of sallow and livid complexion, was admitted into St. George's Hospital under Dr. Chambers, May 6, 1829, with œdema of the lower extremities, cough, dyspnœa and palpitation increased on exertion, starting from sleep, great pulsation of the jugular veins, especially the right, pulse 100 full and strong, urine free but thick.

Had dropsy thirteen years ago. The present symptoms came on three months ago, commencing with cough. Was previously in good general health, and did not complain of shortness of breath.

The *resonance* of the præcordial region is dull over an expanse of five inches in diameter. The *impulse* of the left ventricle is strong, extensive and undulating, with a violent receding jerk or shock when the heart retires. The first *sound* of the left ventricle is scarcely audible, but the second sound is sufficiently smart and loud.

Diagnosis.—Hypertrophy and dilatation of the heart. Hydropericardium. Little if any hydrothorax. Lungs gorged and emphysematous.

℞ Pil. Hydr. gr. v. Scillæ pulv. gr. i. Pil. bis die s. ℞ Haust. nitri, Sp. æth. nitric.—Junip. C. āā ʒi m. ft. haust ter die. ℞ Haust. sennæ, Tr. Jalapæ ʒi m. ft. haust o. m. Diæta lactea.

Five days after admission he was attacked with peripneumony, for which ℥xii of blood were drawn, and ℥x four days afterward. Sputa viscous and rust-coloured, pulse became irregular, sleep disturbed. (Cont. Med.) Was relieved for two or three days, when he had a violent attack of palpitation and orthopnœa. Pulse 110, sputa bloody, mucous râles in the throat and lungs

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judgment and a liberal mind. I have pleasure in stating, in corroboration of the accuracy of my own minutes, that those of Mr. Johnson coincided with them closely and often verbally, though we had no communication until both were written.

(V. S. anodynes and diaphoretics). The paroxysm subsided in 36 hours, but he gradually sank, and died on the 23rd.

Autopsy.—Left ventricle immensely hypertrophous; right, considerably: both, dilated: valves sound: ℥iv of serum in the pericardium: ℥ij in the cavities of the chest. *Lungs.* Hepatization of the inferior lobes on both sides. It is sero-purulent, of chocolate colour, and very flaccid and lacerable. Parts of the middle lobes are in the first degree of peripneumony. The remainder of both lungs is bloated with emphysema and œdema.

Remarks.—The hypertrophy occasioned the power of the impulse, the dulness of the first sound, and the strength of the pulse; while the dilatation rendered the second sound sufficiently loud and smart, the pulse full, and the impulse and præcordial non-resonance more extensive than in hypertrophy alone. The great predominance of the hypertrophy over the dilatation prevented the latter from increasing the first sound. The violence of the back-stroke resulted from the hypertrophy and dilatation conjointly; as the augmented power and weight of the heart, and the increased influx of blood during the ventricular diastole, conspired to render that motion boisterous. The fluid in the pericardium increased the extent of dulness on percussion, and imparted the undulating character to the impulse. The latter is in consequence of the fluid being displaced by the motions of the organ; and as these motions are more violent in cases of hypertrophy, the undulation is proportionably greater. So small a quantity as ℥iv cannot in general be detected with certainty. The pulse, at first regular, as is generally the case in uncomplicated hypertrophy, became irregular in consequence of the engorgement of the heart occasioned by the pulmonary obstruction and the reduction of the vital powers.

As it is almost certain that the hypertrophy existed to a greater or less degree, at the time of his former dropsical attack thirteen years before, and as he had remained during the interval without complaining of bad health, the case tends to show that hypertrophy, in its simple state, may exist for a series of years without creating so much inconvenience as to incapacitate a working man.

Emphysema is one of the most dangerous complications of peripneumony; for, as the dyspnœa which it occasions is liable to be attributed solely to the inflammation, blood-letting may be carried to excess; and thus, the vital powers being diminished while the obstruction in the lungs remains, the patient sinks suddenly and unexpectedly. Several cases of this description have fallen under my observation. The present is not of that number, as the emphysema was detected by Dr. Chambers, and the depletion judiciously regulated accordingly.

Enormous dilatation with hypertrophy of both ventricles; enlarged liver; fits from cerebral congestion; anæmic pulsation, tremor, and murmur of the carotids and subclavians.

Richard Collard, æt. 36, a coachmaker, of large frame, but emaciated and affected with jaundice, was admitted in St. George's Hospital, under Dr. Chambers, August 19th, 1829, with ascites; great œdema of the legs; dyspnœa, exasperated by every movement; cough; great pulsation of the carotids; varicose intumescence and undulation of the jugulars; impulse of the heart preternaturally strong and extensive; pulse bounding but not hard, moderately full and rather vibrating; skin clammy; tongue whitish; bowels open; evacuations of a light clay-colour; urine scanty and deep-coloured. Liver is felt to be enlarged.

Has been more or less ill for two years. Complaint is attributed to fretting. It commenced with shortness of breath and loss of appetite. Dropsy first appeared six months ago, and skin became yellow five or six weeks ago. Is said to be subject to fits.

Auscultation.—Resonance very dull over the whole of the præcordial region. *Impulse* is a powerful heaving, terminating in a jerk or back-stroke: it is felt much more extensively than natural, and in the epigastrium. Both *sounds* are louder, and the first a little more brief, than natural. Above the clavicles there is a slight impulse with very feeble purring tremor and a whizzing sound, not loud or hoarse.

Diagnosis.—Great hypertrophy with dilatation of the heart; enlargement of the liver.

℞ ung. hydr. fort. ʒi hepatis regioni mo. noct. infricand—Haust. nitri, sp. æth. nitrici ʒi m. f^{t}. haust. ter die—Potassæ supertart. ℥ss, jalapæ pulv. gr. x, om. noct. sumend.

During the first week he had three fits, which consisted of stupor, with slight convulsions and stertor, succeeded by sleep. The last attack was of two hours' duration. The dropsy was greatly reduced by the remedies; but he sank exhausted on the eighteenth day after admission.

Autopsy.—*Heart* double its natural size, and, as he was of large frame, it was enormous. Left ventricle would contain a full-sized lemon, and the parietes were three-fourths of an inch thick. Right ventricle was similarly affected, but in a rather less degree. The muscular substance was pale and somewhat softened; it presented a mottled appearance. *Valves* and aorta natural. *Hydrothorax* to four pints. *Lungs* gorged with serum. *Mucous membrane* of the bronchi vascular, and of a dim red colour. *Liver* twice its natural size, of intense yellow (ochre) colour, and its acini were enlarged in every degree up to the size of a pea. *Brain* healthy, but fluid under the arachnoid membrane.

Remarks.—The extraordinary degree of hypertrophy with dilatation was distinctly marked by the extensive dulness of the præcordial region on percussion, without signs of hydropericardium; by the powerful heaving and back-stroke; and by the loudness of both sounds. The predominance of the dilatation over the hypertrophy, prevented the pulse from being so hard and incompressible as the hypertrophy would otherwise have rendered it. Pulsation, vibration and whizzing sound of the larger arteries, as in the present case, are common in anæmia, and still more when an increased quantity of the attenuated blood is transmitted through the vessels with augmented force. They may easily be distinguished from the same resulting from aortic dilatation or disease, by the superior hoarseness of the sound and vigour of the impulse in the latter affections. The disease of the liver was most likely a result of congestion occasioned by impeded circulation of blood through the heart and lungs. The varicose and tumid state of the jugular veins depended on the same cause, while their pulsation was due to the hypertrophy of the right ventricle. The fits were dependent on the violent determination of blood to the brain. I have in many instances known such attacks recur repeatedly, and at last terminate in a fatal apoplectic seizure,—a common result of hypertrophy of the left ventricle. Individuals reduced by years or disease, often sink suddenly, as in the present instance, after the disappearance of much dropsical infiltration. The older physicians supposed that this was in consequence of accumulations in the great cavities; but as, in a large proportion of cases, dissection disproves the existence of such accumulations, dissolution must be ascribed to a failure of the vital powers.

Hypertrophy of the left ventricle, disguised by emphysema; dilatation of both; disease of the interior of the aorta: angina; emphysema and œdema of the lungs.

David Keith, æt. 70, emaciated, of middle stature, and sallow complexion, with a circumscribed redness of the cheeks, was received into St. George's Hospital, September 2d, 1829, subject to severe pain at the inferior part of the sternum and across the epigastrium, which comes on about midnight, accompanied with orthopnœa amounting almost to suffocation. The paroxysm lasts several hours. Cough; dyspnœa on motion; cannot expand either side of the chest; its resonance is good, and in some parts (viz. the anterior and superior) it is more sonorous than natural; pulse 116, large and strong; bowels regular; flatulence.

Says that the asthmatic fits commenced only two months ago; and he attributes them to a "violent cough" which had existed for two months previously. Anterior to that time he did not suffer from dyspnœa. Has taken aperients and been bled.

Auscultation when the circulation was tranquil. *Impulse* of the heart not considerable. Both *sounds* are short and flat, and so loud as to be distinctly audible above the right clavicle. They are obscured below by the catarrhal râles.

Diagnosis.—Dilatation of the heart. Emphysema of the lungs; œdema of the lower lobes; chronic bronchial catarrh.

℞ sp. æth. sulph. ʒss, mist. camph. ʒx, bis die—℞ Hydr. submu. gr. ij, Pulv. Jacobi et Pil. sapon. cum opio āā gr. v. fiant pilulæ iij omni nocte sumendæ.

The asthmatic attacks were diminished for a week, but they recurred with aggravated violence in consequence of his taking fresh cold. October 13th, cough worse, with inability to expectorate from weakness; dyspnœa, emaciation and paleness are creased: voice a whisper; pulse 98, rather unequal; tongue dry; thirst; anorexia; loud sonorous râles over the whole anterior chest. These symptoms increased, and he expired on the 28th October.

Autopsy.—Left ventricle was an inch thick, and dilated to about one-half larger than natural. Muscular fibre red and firm. Right ventricle dilated to the same extent, but not thickened. Margins of the valves in parts slightly thickened with fibro-cartilage, but not sufficiently to cause symptoms. *Aorta*, to the extent of an inch and a half above the valves, very slightly dilated, and, opposite to the origin of the left subclavian, somewhat contracted. Its interior universally overspread with firm, cheese-like matter, intermixed with a few calcareous scales. This state extended to the pelvic bifurcation.

Lungs.—Extremely large, and did not collapse when the chest was opened, in consequence of being universally distended by emphysema and œdema. Air vesicles enlarged—some to the size of pin's heads, and their insufflated state rendered many of the superficial lobules prominent and perfectly pale. Spumous serum exuded copiously on pressure. The lower lobes were in the state of chocolate-coloured flaccid engorgement, heavier than water, but not purulent or lacerable. Some of the great bronchi were of an indelible brownish red colour, and contained purulent mucus.

Remarks.—One of the most instructive features of the present case was, that interposition of the bloated lungs between the heart and the thoracic parietes prevented the resonance of the præcordial region from being so dull, and the impulse of the organ from being so strong, as such a degree of hypertrophy and dilatation would otherwise have rendered them. The action of the heart was not proportionate in violence to the extent of the enlargement, owing, perhaps, to the advanced age and great emaciation and exhaustion of the patient. The existence of murmur from the ruggedness of the aorta could not be ascertained, in consequence of the loudness of the pulmonary râles.

In this, as in many similar cases, the disease of the heart was called into fatal activity by the superadded impediment to the circulation from emphysema and œdema, &c. of the lungs. The supervention of the asthmatic fit during the night was favoured by the recumbent position, and by the accumulation of mucus during sleep. The pain in the region of the heart, commonly called angina pectoris, must be referred to nervous irritation occasioned by the gorged and labouring state of the organ.

Hypertrophy and dilatation from pericarditis; peripneumony.

John Green, æt. 43, a groom, of middle stature and pale, fair complexion, was received into St. George's Hospital under Dr. Chambers, January 6th, 1830, with "a weight in the chest;" dyspnœa and palpitation increased by any exertion; cough; viscid, rust-coloured sputa, sometimes black with grumous blood; hoarseness; orthopnœa; pulse 120 sharp; tongue thickly furred and yellow in the centre, pale at the edges; thirst; anorexia; bowels costive; urine scanty and offensive; emaciation.

Had been suddenly attacked, three months before, with dyspnœa, palpitation and the other symptoms (pericarditis). They had been occasionally relieved, but, on his admission, were worse than ever. Previous to the attack, he was healthy.

Auscultation.—Increased sound and impulse of the heart.

Diagnosis.—Peripneumony; enlargement of the heart.

V. S. ad ℥xij. ℞ Haust. salin. cum oxym. scillæ ʒss, 6tis horis. ℞ Hydr. submu. gr. v. hac nocte, et haust. sennæ cras mane. Diæta parciss. The blood was highly buffed; and as the symptoms continued and the pulse had become 84 and full, venesection was repeated to the same extent, and he took calomel gr. ij with opium half a grain, 6tis horis. These and all the other means employed were incapable of affording relief, and he expired on the sixth day.

Autopsy.—About ℥xij of serum in the cavities of the pleura; old adhesions on the left side; lungs more voluminous than natural from emphysema and œdema; the inferior portions of both were hepatized; namely, of reddish chocolate colour, heavier than water, flaccid, lacerable, and in some parts purulent. Where the latter character exists, the colour is paler and the ramollissement greater.

Pericardium partially overspread with organized lymph, but not adherent. Left ventricle three fourths of an inch thick at the base and one half at the apex; its cavity dilated to twice its natural capacity. Right ventricle equally dilated, but not hypertrophous. Lining membrane on both sides was stained of deep crimson colour. Muscular substance was pale and flaccid, but not lacerable. Valves natural; a slight steatomatous deposition around the coronary arteries.

Remarks.—The lymph on the pericardium, the paleness and flaccidity of the muscular substance, the intense redness of the lining membrane, and the sudden supervention of all the symptoms three months previous to admission, afford almost positive proof that the affection was originally pericarditis or endopericarditis, the softening occasioned by which had led to the great and rapid dilatation. The increased action and sound of the heart, and the state of the pulse, denoted the enlargement of the organ. The supervention of peripneumony while the heart was still labouring under the effects of inflammation, rendered the case extremely formidable, if not altogether hopeless.

Enormous hypertrophy and dilatation of the heart; disease of the aortic valves with regurgitation and jerking pulse; universal adhesion of the pericardium; acute rheumatism; anæmia.

John Copas, æt. 24, a gardener, of middle stature and robust frame, cadaverously pale, was admitted into St. George's Hospital under Dr. Chambers, October 14, 1829, with universal rheumatic pains, aggravated when warm and perspiring; very slight œdema of the legs; face rather puffy; palpitation; sleep disturbed by starting; the pulsation of the heart not only perceptible to the touch, but visible over nearly the whole anterior surface of the chest, and particularly in the epigastrium. Resonance of the præcordial region extremely dull; pulse 120, full, strong and regular, but *compressible*—a circumstance particularly pointed out to me by Dr. Chambers.

He had suffered from acute rheumatism eight years before, and had never since been exempt from palpitation.

Auscultation was not employed.

Diagnosis.—Acute rheumatism; organic disease of the heart; adhesion of the pericardium.

℞ Pil. Hydr. gr. iij, pulv. scillæ gr. ij, pulv. digitalis gr. ss, ter die—℞ Inf. aurant. c. ʒx, Sp. æth. nit. et sp. junip c. āā ʒi, T^{r}. hyoscyami m x, 6^{tis} horis. ℞ Haust. sennæ, pulv. Jalapæ gr. vi, potassæ supertart. ʒi, alterno die.

He died within twenty-four hours, after an attack of hæmoptysis to a considerable extent.

Autopsy.—Universal adhesion of the pericardium. The layer of lymph, forming the medium of adhesion, was thin and dense. The heart was judged to be nearly three times its natural dimensions. The enlargement was principally in the left ventricle, the walls of which were an inch and a half thick, and the cavity larger than the largest orange. The right ventricle was similarly affected, but in a less degree. The *aortic valves* were thickened, nodulated and corrugated by an opake, yellow degeneration, partly cartilaginous and partly steatomatous. This had caused the detachment of the angular extremities of the valves from their insertions; so that, being adherent by their centres only, they projected loose into the artery, and were destitute of fulcra by which to oppose the reflux of blood from the aorta.

Remarks.—The degree of enlargement which existed in this case is seldom exceeded. There is little doubt that the affection originated in the attack of rheumatic endopericarditis eight years before, by which adhesion of the pericardium and the disease of the valves had been occasioned—lesions that never fail to induce more or less dilatation, and generally hypertrophy. As the dilatation was so enormous, it is not improbable that softening from the pericarditis contributed in the first instance to its production. The thinness and density of the lymph indicated the oldness of the adhesion; for in recent cases the deposition is always soft, and often several lines in thickness. The adhesion was inferred from the obvious hypertrophy with dilatation, the antecedence of rheumatic pericarditis, and the remarkable movement in the epigastrium, probably occasioned by retraction, as supposed by Dr. Sanders. This is one of the very few cases in which I have observed this phenomenon, nor can I say that the retraction was very distinct even here. Did regurgitation of the aortic blood consequent on the disabled state of the valves, occasion the compressibility of the pulse, noticed by Dr. Chambers? This question, written in 1829, I was soon after able to answer in the affirmative, having fully ascertained that the eminently *jerking* pulse (which, though full and strong, is always compressible) is the characteristic pulse of aortic regurgitation (see p. 379). At the time when this case was written, I was doubtful whether the pulse was referable to the adhesion of the pericardium or to the regurgitation, as I had always met with it in the conjoint affection.

It is manifest that regurgitation must have a powerful effect in producing enlargement of the left ventricle; for the whole weight of the arterial circulation, instead of being sustained partly by the valves, rests constantly, and exerts its expanding force, upon the ventricle.

The hæmoptysis depended on the state of the heart. For, as the retrograde pressure of blood in the left ventricle had precisely the same effect as a valvular obstruction in opposing the passage of blood from the lungs through the left side of the heart; while, at the same time, the right ventricle, hypertrophous and dilatated, expelled a preternatural quantity of blood with augmented impetuosity, the delicate vessels of the lungs, exposed to these conjoint forces operating in opposite directions, yielded to the pressure, and relieved themselves by transudation of blood into the air passages. It is for this obvious reason that pulmonary apoplexy and hæmoptysis are more frequently found connected with an impediment on the left side of the heart, and simultaneous hypertrophy of the right ventricle, than with any other lesions of the organ.

Dilatation of the heart; hydropericardium; hydrothorax.

John Snowden, æt. 38, tall, thin, sallow, with circumscribed redness of the cheeks, was received into St. George's Hospital May 19th, 1829, with orthopnœa; excessive dyspnœa on the slightest exertion; cough; watery expectoration; ascites; face puffed and leucophlegmatic; great œdema of the legs; undulating or rolling motion in the præcordial region; pulse 110, weak and irregular; urine scanty.

He had long been short-winded, but to no considerable degree until two months before admission, when he was suddenly attacked with excessive dyspnœa while walking. This symptom increased, and in a fortnight was followed by dropsy.

Auscultation.—Resonance dull over an unusual extent of the præcordial region. *Impulse* of the left ventricle is slightly increased, but *undulating* and *not synchronous with the ventricular contraction* as indicated by the first sound. Over the right ventricle the impulse is weaker. *Sounds*, are short, flat, and audible over the whole anterior surface of the chest. Neither coincides with the radial pulse, and they are so much alike as to be with difficulty discriminated.

Diagnosis.—Dilatation of the left ventricle, without attenuation of its parietes; hydropericardium; hydrothorax (ascertained by percussion.)

V. S. ad ℥x—℞ Elaterii gr. i, calomel. gr. ij, cras mane.—Potûs supertart. potassæ lb. i in die. He improved considerably for a fortnight, when he was bled to ℥x for increase of cough and slight erysipelas of the face. These were mitigated, but the debility and dropsy increased. Pulse 120 (Haust. sulph. quinæ ter die.—℞ Haust. opiat., oxymel scillæ et sp. æth. nit. āā ʒss omni nocte.) In a fortnight the cough and dropsy were greatly diminished, and he was able to leave his bed a little.

Auscultation.—The impulse coincides better with the ventricular systole, and the heart is more distinctly felt to strike the ribs. Pulse is still weak, and not perfectly synchronous with the ventricular contraction. *Diagnosis.—Hydropericardium diminished.* Emaciation and debility now made rapid strides, the dropsy began to re-accumulate, and in another fortnight he sank.

Autopsy.—Upwards of Oij of serum in the pleura; ℥iii or iv of bloody fluid in the pericardium. *Lungs.* The left was healthy above, but the inferior lobe was gorged with blood, and heavier than water. The right contained some suppurating tubercles. *Heart.* Left ventricle was considerably dilated, and its parietes were half an inch thick. The right ventricle was rather less dilated than the left, and its parietes natural, or thinner if either. *Valves* healthy. The apex of the left ventricle contained a polypus which had softened or suppurated in its centre; and roundish nodules of lymph were found in the interstices of the columnæ carneæ.

Remarks.—The short, flat and loud first sound, and the weak and irregular pulse, indicated the dilatation; while the rather increased action of the left ventricle, though partly attributable to the accelerated and disturbed state of the circulation, denoted that the parietes were not attenuated.

In cases of dilatation, when the general constitutional powers are still tolerably good, and the increased capacity of the heart does not greatly predominate over its muscular strength, the pulse is generally regular, and, though soft, it has frequently a considerable degree of fulness. But when the strength fails, as in the present instance, or the heart is otherwise excited beyond its contractile power, the same pulse may become both weak and irregular. The latter character, therefore, must be regarded rather as incidental, than essential to dilatation.

It may be inquired how, in this case, the pulse was weak, while the action of the left ventricle was increased. This apparent anomaly is of frequent occurrence; and, what is still more remarkable, it may take place in cases of hypertrophy as well as of dilatation. In fits of asthma or great dyspnœa, for example, the pulse is often scarcely perceptible, while the heart is felt to be in a violent tumult. In other cases, both the impulse and pulse are diminished, and nothing is then felt in the præcordial region, but an obscure, profound, rolling or fluttering motion.

The inference from these facts appears to be, that when the heart is congested beyond its propulsive power, its efforts are expended on itself, without communicating strength to the pulse; and that when the engorgement is extreme, its muscular power is more or less paralysed or suspended.

In addition to its other qualities, the pulse, in the above case, was later than the ventricular systole. I have found this to occur in nearly all conditions of the heart in which the blood was propelled with difficulty, but especially in dilatation, and in contraction of the mitral valve.

The hydropericardium was indicated by the undulatory nature of the impulse; by its want of coincidence with the sound of the ventricular contraction; by the sensation, communicated through the stethoscope, that the heart did not strike the ribs *immediately*; and by the extensive dulness of the præcordial region on percussion. These, according to my experience, are the best physical signs of hydropericardium; and, when supported by general signs, they will rarely, I believe, be found fallacious.

The polypi evince the difficulty with which the blood was circulated through the heart, these formations commonly being results of its stagnation. Their organised appearance and internal softening afford reason to believe that they had existed for a considerable time. The case presents an instance of failure of the vital powers on the disappearance of dropsy,—a common event in aged persons, or exhausted constitutions.

The bleedings practised were injudicious, as he was already too anæmic and emaciated to bear them. Accordingly, the pulse rose, and the debility and dropsy increased.

Simple dilatation of the heart, with softening, producing a feeble, irregular pulse; pleurisy.

Patrick Gillan, æt. 43, a hawker, admitted into St. George's Hospital, under Dr. Chambers, June 24, 1829. Pain in the left hypochondrium with inexpansibility of the ribs over the part affected; slight cough; scanty, white expectoration; decubitus easiest on the side affected; pains in the head and shoulders; orthopnœa; difficulty of respiration increased by ascending; pulse 80, feeble, irregular, and extremely intermittent; skin cool; tongue furred and yellow; bowels costive; urine high-coloured. Turgescence without pulsation of the jugulars.

A fortnight before admission he was seized with general rheumatic pains, stitch in the left side, and dry cough. During the preceding winter he had vomited two quarts of black blood, intermixed with food.

(V. S. ad ℥xij—℞ Haust. salin: cum sulph. potassæ, ʒi, 4^{tis} horis. Diæta parciss.)

Auscultation.—No *impulse* that raises the head, (applied to the cylinder,) but a fluttering motion, with an occasional shock of some strength. A short, flapping first sound, not much louder than natural. No bellows-murmur of either sound.

Diagnosis.—Passive dilatation of the heart; no ossification of the aortic valves, nor disease of the mitral according to the evidence of auscultation, though the irregular, fluttering action, *and feeble, intermittent pulse, favour the idea of regurgitation into the left auricle.*

Œdema of the legs with scanty urine supervened. Diuretics and purgatives with camphor and hyoscyamus were prescribed, and the emplast. belladonnæ was applied over the heart. The effect of the medicines was satisfactory, but the constitution was worn out, and he died a month after admission.

Autopsy.—The heart was dilated to nearly double its natural size; the parietes were about natural, or attenuated if either, but they were very flabby, and had externally a leucophlegmatic or infiltrated appearance and feel. *Valves* and aorta were sound. *Lungs* were gorged with blood and serum, and contained a few isolated tubercles. The surface of the inferior lobe on the left side, and the corresponding extent of the pleura costalis, were covered with old, whitish lymph, which by its adhesions formed a sac, enclosing a pint and a half of serum.

Remarks.—The feeble and intermittent pulse in this case led some to suppose that there was disease of the valves. The case itself (and it is by no means a solitary one) proves that such a pulse may exist totally independent of valvular disease when the debility of the heart or of the constitution is very great.

The softened state of the organ contributed to render its action more feeble and irregular, and its sounds weaker, than might otherwise have been anticipated from such a degree of dilatation. I have shown in the chapter on softening that it is the attribute of this disease to produce these effects (see p. 339). Turgescence without pulsation of the jugular veins, as in this case, is very characteristic of a dilated or otherwise enfeebled right ventricle. When not enfeebled, and especially when hypertrophous as well as dilated, the turgescence is always accompanied with pulsation. As congestion of the venous capillaries predisposes to hæmorrhage, it is probable that the hæmatemesis which occurred during the previous winter, was attributable to this cause. The death of the patient was accelerated by the pleurisy, and not only by its direct effect, but by its hurrying the action of the heart, and increasing the embarrassment of the circulation. It is, indeed, generally by accidents of this kind that diseases of the heart are brought to their fatal termination; and this fact suggests an important practical lesson—

that, in persons affected with organic disease of the heart, all complaints capable of hurrying the circulation, and especially those of an inflammatory nature, should be regarded and treated as maladies of serious importance, capable of suddenly and unexpectedly producing a series of the most dangerous effects.

Enormous dilatation and extreme attenuation of the left ventricle; dilatation and hypertrophy of the right; pulmonary apoplexy; enlargement of the liver.

William Lambert, æt. 52, an eating-house keeper, tall, emaciated, of exsanguine, sallow complexion, was admitted into St. George's Hospital, under Dr. Chambers, September 6, 1829, with pain in the chest, principally at the base of the sternum, and increased by full inspiration. Cough; expectoration copious, viscous, deeply coloured with blood; dyspnœa with cough in agonising paroxysms, induced by any exertion, particularly ascending; the right jugulars slightly tumid, with pulsation; fluctuation of the abdomen; slight œdema of the legs; enlargement and induration in the region of the liver; decubitus easiest on the right side. Pulse 70, intermittent, rather weak, sometimes scarcely perceptible: skin cool, tongue furred, of cream colour; bowels costive; urine high coloured and scanty.

Ill nine months. Complaint began (after protracted mental anxiety) with cough and dyspnœa, which frequently occurred in paroxysms. The ascites had existed, more or less, for two or three months previous to admission; and the œdema of the legs for a week only.

Auscultation.—The inferior dorsal region of the chest, on the right side, is dull on percussion, and has a slight crepitant râle. The superior lobes of the lungs are resonant, but the respiratory murmur is puerile and bronchial. The *impulse* of the heart is slightly tumultuous or confused, but very feeble. *Sounds* are little louder than natural, but the first is short, like the second: they are audible at the clavicles, especially the right.

Diagnosis.—Peripneumony or pulmonary apoplexy of the right lung; bronchitis; dilatation of the heart; (particularly the right ventricle?) enlarged liver.

Cucurb. cruent. inter scapulas ad ℥xij ℞ Inf. Rosæ ℥iss, magnes. sulph. ʒij, sp. ætheris nitric. ʒss, 6^{tis} horis.—℞ conf. sennæ ʒi, potassæ supertart. ʒss, omni nocte. Diæta lactea. The symptoms were alleviated at first; but effusion was found, by auscultation and percussion, to increase rapidly in the right pleura, and the cough and dyspnœa suffered a corresponding aggravation. When the circulation was accelerated, the action of the heart was occasionally found to be more vigorous than natural, though the pulse, at the same time, continued feeble and small, but tolerably regular. The sputa maintained their deep muddy red stain to the last. Orthopnœa with the utmost distress from a sense of suffocation became constant, and the patient expired five weeks after admission.

Autopsy.—The right cavity of the chest was filled with clear, chlorine-coloured serum; and the lung, compressed against the spine, was reduced to the size of a spleen. The pleura pulmonalis was covered with lymph, in honeycomb reticulations; and the pleura costalis was mottled with patches of red vascularity. The compressed lung felt doughy and non-crepitant. The margin of the lower lobe was in the second degree of hepatization, bordering on the third, a little pus exuding on pressure. In the midst of this was a mass of pulmonary apoplexy, as large as an egg, claret-coloured, granular, of great density, and bounded abruptly by a wall of straw-coloured lymph. Similar masses existed in the other lung, with sanguineous engorgement, but no hepatization. *Heart.* The *left ventricle* was dilated to a capacity which would easily contain the largest orange, or even a small melon. The parietes did not anywhere exceed a quarter of an inch in thickness, and throughout the lower half they varied from one to two lines. Over a small extent, near the apex, the muscular substance was totally deficient, and the membranes alone formed the barrier. At this part, however, the pericardium had been thickened and strengthened by an external layer of lymph,—as takes place over large vomicæ contiguous to the pleura, and which we cannot but regard as a wonderful provision of the Author of nature to obviate sudden death, which must otherwise so frequently occur. Many large coagula of bloody fibrine lined the cavity, and adhered tenaciously to the columnæ carneæ. The *right ventricle* was dilated, but to a rather less extent than the left, and its parietes were in parts four or five lines thick. Both *auricles* were dilated. *Valves* were all sound. *Aorta* was slightly dilated, but otherwise healthy.

Remarks.—The feebleness of the heart's action, the brevity of the first sound, the weakness of the pulse, and the general symptoms of venous retardation, indicated the dilated and debilitated condition of the heart. The occasional intermissions of the pulse, and the somewhat tumultuous or confused nature of the impulse, might have led to a suspicion of disease of the mitral valve and regurgitation into the auricle: but in such cases the pulse is not only intermittent, but unequal and irregular, and it becomes remarkably so towards the fatal termination. These, however, were not its characters in the present instance; and as there was not, moreover, any bellows-murmur accompanying the sounds, valvular disease was excluded from the diagnosis. A sufficient cause for the intermittence and unsteady impulse existed in the extreme degree of dilatation, and the consequent labour of the heart to propel its unnatural burden. The increased impulse of the heart, when the circulation was accelerated, proceeded from the thickened state of the right ventricle; and to the same circumstance, together with the retardation in the left ventricle, is the pulmonary apoplexy to be attributed. This affection was indicated by the crepitant râle and the bloody sputa. It was the latter, however, which formed the diagnostic sign; for the stain of blood was redder, and persisted more unchanged to the last, than occurs in peripneumony, in which affection, the sputa, though pinkish at first, soon became rust or fawn-coloured, and even this stain gradually decreases as the disease advances to its resolution, or degenerates into purulent infiltration. When, therefore, such a state of the expectoration as existed in the present case, accompanies signs of obstruction on the left side of the heart, especially if conjoined with those of increased action of the right ventricle, pulmonary apoplexy may be anticipated. The tenacious coagula adherent to the left columnæ carneæ evinced that the circulation through the ventricle had been languid. These formations, when they choke the cavities, cause a remarkable aggravation of dyspnœa, and by this they may often be recognised for a week, or even longer, before death, the patient having a constant agonizing feeling of imminent suffocation. The enormous size of the left ventricle caused it to occupy a more central situation than natural. Its sounds, therefore, being more audible at the base of the sternum than in the left præcordial region, the dilatation was supposed to be greater on the right than on the left side. Laennec experienced and pointed out this source of fallacy (tom. ii. 507). Its rarity renders it unimportant. The pulsation of the jugulars is to be referred to the hypertrophy of the right ventricle; and the enlargement of the liver found its origin in congestion resulting from impeded circulation through the heart.

Hypertrophy and dilatation of both ventricles; dilatation of the aorta; roughness of its interior; bad effects of excessive blood-letting.

Henry Macearl, æt. about 45, an old soldier, tall, meagre, sallow, and livid when cold, was received into St. George's Hospital, October 28, 1829, with orthopnœa; impulse of the heart stronger, lower, and more extensive than natural; occasional pain in the left side, when attempting to lie upon it; somnolency; languor; no dropsy ever; pulsation of the carotids; pulse 116 —a jerk, followed by full, strong and vibrating tension,—regular; tongue white; bowels open.

Eighteen months previous to admission he received a kick from a horse on the præcordial region, which gave rise to his complaint.

Auscultation.—Resonance of the præcordial region is rather dull: that of the chest elsewhere, good. *Impulse*, very powerful above the clavicles, especially opposite to the arteria innominata. It is accompanied with purring tremor, and a loud, hoarse, abrupt bellows-murmur, which, when traced downwards along the sternum, becomes more hissing, and, as it were, superficial. It retains the same characters, though somewhat stifled, in the præcordial region, where it drowns the natural sound of the ventricular contraction. The *impulse of the heart* is much stronger than natural, and is followed by a vigorous back-stroke.

Diagnosis.—Hypertrophy and dilatation of the heart: dilatation of the aorta; disease of its internal coat, from the aortic valves to beyond the arch. Fiat V. S. ad ℥x, ægro recumbente. ℞ T^{r} opii m xxx, mist. camph. ʒx. Diæta lactea.

In the course of ten weeks he was sparingly bled six or seven times, as he stated it to be "the only thing that afforded him relief." He also took various various formulæ of opium, æther, Infus. Digitalis, Ext. Lactucæ, and aromatics; but they had little effect, and he progressively declined. Œdema of the lower extremities supervened, with constant orthopnœa; pain in the præcordial region; ghastly paleness, without lividity, of the face; frequent paroxysms of dyspnœa; and extreme anxiety and distress. He died Jan. 15, 1830.

Autopsy.—The left ventricle was three-fourths of an inch thick, and its cavity was dilated to one half more than its natural size. The right ventricle was equally dilated, but only slightly thickened. *Valves* were healthy, except that the aortic were a little cartilaginous, but perfectly flexible. *Aorta* was somewhat dilated; and the whole of its inner surface, from the valves to beyond the arch, was rendered extremely rough by steatomatous or cheese-like degeneration, deposited in great abundance. Patches of the same were found as low as the pelvic divarication.

Remarks.—The violence of the heart's action, and the strength of the pulse, left no doubt of the hypertrophy: and the dilatation was denoted by the extent of the impulse, the deficient præcordial resonance, and the fulness of the pulse. The dilatation and roughness of the arch of the aorta were indicated by the impulse, sound, and tremor above the clavicles; and the ascending aorta was presumed to be in the same state, from the existence of the same sound along its course, only more hissing from the greater contiguity of the artery to the ear, and the interposition of a less resonant medium It is apparent from this case, that a murmur generated in the aorta, may extend to the heart and obscure its sounds. Caution is, therefore, requisite not to mistake it for a result of valvular disease. The diagnosis is given at p. 384.

The vigorous back-stroke is to be remarked as a concomitant of hypertrophy with dilatation; the purring tremor of the pulse, as a consequence of powerful propulsion of the attenuated blood through a rugged aorta: and the extreme severity of the dyspnœa, as a result of the complication of disease of the aorta with that of the heart, in an asthmatic subject. This was, in short, one of the worst forms of cardiac *asthma.*

The case strongly exemplifies the bad effects of excessive blood-letting. He was bled six or seven times in ten weeks: the result was, ghastly paleness (anæmia), dropsy and progressive decline.

Great hypertrophy with dilatation of the left ventricle; ossification of the aortic valve; chronic pericarditis with effusion; hemiplegia and apoplexy.

Richard Porter, æt. 52, a cook, of small stature, pale, emaciated, was admitted into St. George's Hospital, under Dr. Hewett, April 8th, 1829, with hemiplegia of the left side; mouth distorted to the right, but partial paralysis of both sides of the face; a sensation of fulness and tightness about the inferior part of the sternum; cough; starting from sleep in a fit of palpitation and suffocating asthmatic dyspnœa; anasarca; pulse 96, full, and tolerably firm and regular.

Ten years before admission he had apoplexy and hemiplegia of the left side, which disabled him for half a year. He then resumed his work as a cook, and prosecuted it until three weeks ago, when he took cold, and became affected with anasarca, to which he had been subject. With this account of the early history I was favoured by Dr. Hewett, under whose care the patient was admitted. I did not see him till July 2.

Auscultation, three months after admission. Very loud rasping murmur. (A momentary examination.) *Diagnosis. Disease of the valves of the heart.* He died eight days afterwards, in consequence of a fit of apoplexy.

Autopsy.—Head. A small coagulum of blood under the dura mater, at the vertex of the brain, and three or four ounces of serum at the base. *Chest.* In the cavities of the pleura were upwards of three pints of serum; and in the *pericardium* was above a pint, deeply coloured with blood. The whole interior of the sac, and the surface of the heart, were invested with a thick stratum of shaggy, and highly vascular, reddish lymph. *Heart.* The left ventricle was thickened to almost double—or to nearly an inch; with general dilatation of the heart. *Aorta.* Its internal membrane was slightly corrugated by steatomatous degeneration, intermixed with a few calcareous scales. *Valves.* The edge of one of the aortic valves was encumbered with an osseous concretion as large as a pea, of an elongated form, projecting into the artery, and with an irregular, denuded and scabrous surface.

Remarks.—Though the details of this case are defective, it is, notwithstanding, one of great practical value. It demonstrates that a very considerable impediment in the aortic valves does not necessarily prevent the pulse from being full, and tolerably firm and regular, the reverse of which was believed by the old writers, particularly Corvisart, who has been followed by Louis, Bouillaud and almost all other authors (see p. 378). It shows that a scabrous ossification occasions not only a loud murmur, but one of a rasping or grating character. The case, furthermore, presents one of the numerous instances of palsy or apoplexy connected with hypertrophy of the left ventricle; and, as an interval of ten years had elapsed between the first and second paralytic attack, during which he had continued at his accustomed avocations, it shows with what an extent of disease of the heart the functions of life may be maintained. Steatomatous and calcareous disease of the aorta is so frequently accompanied with hypertrophy of the left ventricle, that it is natural and rational to regard the latter as a result of the obstacle to the circulation presented by the former. But, on the other hand, the frequent occurrence of the same disease in the arteries of the brain when the left ventricle is hypertrophous, leads to the inference that over-distention may occasion it, (see p. 259,) and, consequently, that its existence in the aorta may sometimes be secondary to the hypertrophy of the ventricle. On either view, the diseases described, of the aorta and of the heart respectively, are cause and effect, and hence, the practical deduction is, that, when either exists, it is requisite to keep the circulation tranquil, in order to prevent the development of the other.

The chronic pericarditis probably took its date from the attack of cold three weeks before his admission, and occasioned the sensation of fulness and tightness about the inferior part of the sternum. It is not unusual to find bloody fluid effused by organized lymph of the pericardium, especially when, as in the present instance, this membrane is in a state of chronic inflammation.

Simple hypertrophy; contraction of the aortic valve to the size of a small pea; asthmatic fits about noon daily.

Wm. Hedgley, æt. 10, was admitted into St. George's Hospital, under Dr. Hewett, April 17, 1830, with respiration very hurried; temporary pain and constriction in the præcordial region; extensive pulsation of the heart; slight cough; œdema around the eyes; daily febrile accessions with palpitation, coming on about noon, and consisting of chilliness for an hour, heat for half an hour, and perspiration till evening; pulse 120, very small, weak and unequal; tongue thickly furred, moist; skin cold, perspiration: bowels regular; urine scanty, dark and thick.

Did not complain until seven weeks ago, when the paroxysms, accompanied with pain at the heart, first attacked him (endocarditis?).

Auscultation.—Resonance of the chest natural. Impulse of the heart increased. Sound of the ventricular contraction is that of sawing (bruit de scie). He died three weeks after admission, viz. May the 11th. I was favoured with the notes of this case by Dr. Hewett, as I did not see the patient until the post-mortem examination.

Autopsy.—Walls of the *left ventricle* were upwards of half an inch thick, and very firm; those of the *right* were slightly thickened; both cavities were about natural. The *aortic aperture* was contracted by fibro-cartilage to the size of a small pea. Two ounces of serum in the pericardium, and six in each pleura. Lungs, at the lower parts, were congested and somewhat condensed.

Remarks.—The disease of the valve was clearly indicated by the sawing-murmur, and the hypertrophy by the increased impulse. The case proves that an extreme degree of contraction of the aortic valves renders the pulse small, weak and unequal; while the preceding case proved that a moderate degree had not that effect. The valvular contraction was manifestly referable to the endocarditis ten weeks before death, and the case displays how rapidly so serious a lesion may be occasioned by inflammation.

Why the intermittent febrile paroxysms occurred at the same hour daily, is not very apparent, unless the patient had been under the influence of malaria, which I could not ascertain. In the case of May, the paroxysm occurred at the same hour every night.

Dilatation of the heart: ossification and slight dilatation of the ascending and descending aorta; dilatation of the bronchi; hydrothorax; ossification of the cerebral arteries.

Richard Storer, æt. 73, feeble and decrepit, was received into St. George's Hospital, July 8th, 1829. His symptoms were, palpitation; dyspnœa, aggravated by the slightest exertion; respiration extremely wheezing; cough; copious expectoration; universal dropsy; jugular veins turgid without pulsation; pulse 90, full, strong, and tense.

Subject to a chronic cough for fourteen years. Swelling of the face came on ten weeks before admission, and was followed by that of the feet, scrotum, &c.

Auscultation.—Slight pulsation and soft bellows-murmur above the clavicles; *impulse* of the heart not perceptible to the hand, and it can only be felt occasionally by the cylinder. Its power is then considerable, but it is rather a blow, than a heaving of the thoracic parietes. *Sounds*—both are short and flat; neither is very loud, but the second is the louder. Excessive mucous râles in the chest, which obscure any murmurs of the heart. (Bruit de soufflet was distinguished at a subsequent examination by Mr. Johnson.) At the lower part of the left scapula there is loud pectoriloquy and gurgling râle.

Diagnosis.—Dilatation of the heart; no aneurism, nor appreciable dilatation of the arch of the aorta. Hydrothorax and dilatation of the bronchi on the left side.

℞ pil. Hydr. gr. iij, pulv. scillæ gr. i. pil. ter die sum.—℞ potus potassæ super-tart. Oj quotidie—℞ elaterii gr. ss, hydr. submuriat gr. ij, alterno quoque mane sumend.

In three weeks the dropsy was greatly reduced, but as the legs continued œdematous, slight incisions were made with the lancet in the calves, by which the fluid was evacuated, and a considerable quantity of blood lost. After this, his strength gradually failed, and he sank in four days.

Autopsy, for the account of which I am indebted to Mr. Johnson.—Heart was very large. All its cavities were dilated. The parietes of the left ventricle were about natural, or perhaps thicker. *Valves* healthy; but there were slight calcareous depositions beneath the bases of the aorta, and under the internal membrane of the heart, between the aorta and the mitral orifice. *Aorta.* No dilatation of the arch; but some in the ascending portion, immediately before the branches; and again, beyond the origin of the left subclavian. Osseous depositions, underneath the lining membrane, were scattered generally throughout the aorta and great branches; and at the mouth of the left subclavian, a denuded patch was found. *Brain.* The arteries were diseased; especially the basilar, which was very large and rigid. *Lungs.* The left cavity of the chest contained upwards of a pint of fluid; and the lung, compressed and collapsed, was imperfectly crepitant, and so dense as to sink in water. This condition was most marked, opposite to the inferior half of the scapula; to which part, and above, the lung was inseparably adherent. The bronchus entering the portion of lung, divided into many large branches; all of which were drawn, by the adhesion of the pleura, into close apposition with the thoracic parietes; and one, not larger than a writing quill, was dilated at its extremity to the dimensions of a small nut. The *left lung* was œdematous above, and congested with blood below.

Remarks.—The signs of dilatation were, the feeble impulse, and the short, flat sound of the ventricular systole. The more vigorous impulse occasionally felt, and the strength and tension of the pulse, indicated that the muscular power was still considerrable: in other words, that the walls of the left ventricle were not attenuated. In a young and robust subject, such a heart produces increased impulse, as in the case of Dolan. The remarkable wheezing of the respiration, led the attending physician and others to the suspicion of aneurism or of great dilatation of the *arch* of the aorta, these affections sometimes producing that symptom by pressure upon the trachea. It was in reference to this opinion that I gave an opposite one in the diagnosis. The contra-indications were, the want of strong pulsation, purring tremor, and loud rasping sound above the clavicles. The slight impulse and murmur which existed there, were owing, the former, perhaps to the throbbing of the subclavians; the latter, to the ossification of the interior of the aorta and the dilatation below the innominata. The dilatation of the ascending aorta might have been recognised by tracing the murmur down the sternum, had not the loudness of the pulmonic râles rendered this impossible. Dilatation of the bronchi was inferred, because, as he exhibited no signs of phthisis, the pectoriloquy and gurgling râle could not be attributed to vomicæ. The idea was, further, countenanced by his having been subject to a chronic, asthmatic cough for fourteen years, when at an extremely advanced age: circumstances peculiarly favourable to the production of bronchial dilatation. Disease of the cerebral arteries may here be remarked as accompanying enlargement of the heart and ossification of the aorta. The ossifications I should ascribe to his advanced age. The effects of the elaterium were good; but it is a remedy which cannot be given with impunity to subjects so old and enfeebled as the present, without constant watching and great discretion on the part of the practitioner.

After rheumatic endocarditis, dilatation of all the cavities, with natural thickness of the parietes; vegetations of the left auricle and mitral valve, causing regurgitation; superior cuspis of the mitral valve across the aortic orifice; contraction of the aorta.

John Dolan, æt. 28, a servant, of robust frame and pale, delicate complexion, was admitted into St. George's Hospital, under Dr. Chambers, May 27, 1829, with palpitation, increased on exertion; orthopnœa; cough; thick, white sputa; decubitus on either side; undulation of the jugulars; slight œdema of the legs; pulse 110, small, and very weak; bowels regular.

Five weeks before admission, he took cold while travelling, and was seized with pain at the heart, and cough (endocarditis?). He was bled, and a few days ago cupped, with relief. Œdema has only existed a week. Had rheumatic fever two years ago, and several times previously.

Auscultation.—Resonance of the præcordial region, dull over a very large extent. *Impulse* much stronger than natural, and felt far beyond the usual limits and in epigastrio. *Sounds* are louder than natural; especially the second in the left præcordial region: the first is remarkable for a strong, but not grating bellows-murmur, most distinct on the left side.

Diagnosis.—Hypertrophy and dilatation of the heart; dilatation of the left auricle; obstruction, probably cartilaginous, in the aortic orifice. Emplast. Lyttæ regioni cordis.—℞ Haust. salin., Tʳ Hyoscy. ʒss, 6ʳⁱˢ horis.—Diæta lactea. He subsequently took, in various formulæ, calomel, haust. sennæ, sp. æth. nitric, Tʳ Digitalis, et acet. potassæ. The emplast. opii was applied over the heart. But, in a fortnight, the œdema and ascites had made progress; and in another week he was confined to bed, with constant drowsiness and profuse perspiration, which, in two days, were followed by extreme intumescence of the face. These symptoms persisted five or six days more, when he became incoherent, stupid, and, finally, comatose; in which state he expired, June 29th.

Autopsy.—Both ventricles dilated. Walls of natural thickness. Both auricles also dilated; the left to more than double, and its interior is covered, over an extent of two square inches, with small cauliflower vegetations. These likewise pervade the whole of the mitral valve and the chordæ tendineæ, rendering the margins of the valve so thick and knotty, as to prevent them from closing accurately. The closure is further impeded by contraction of the chordæ The right cuspis of the valve is displaced in such a manner as to extend across the aortic orifice and obstruct the egress of the blood. Mitral orifice, from the auricular side, expands perhaps too widely in consequence of the dilatation of both cavities. *Aorta.* Valves healthy, but the artery is remarkably contracted throughout, and, half an inch in front of the left subclavian, it is corrugated. *Lungs* œdematous and gorged with blood. Two small portions intensely dark, granular, and so dense, as to sink quickly in water (pulmonary apoplexy). *Brain* contained an ounce of serum; and *pericardium*, half an ounce. *Kidney*, large and pale.

Remarks.—This case proves, that if dilatation be accompanied with a natural thickness of the parietes, it produces the symptoms of hypertrophy: viz. increased action. This holds true, however, only in reference to young or robust subjects,—not to the old, or otherwise enfeebled (as Störer). The great degree of the enlargement was indicated by the extent of the impulse, and of the dulness on percussion.

The murmur which attended the ventricular contraction, was occasioned, not only by the position of the cuspis of the mitral valve across the aortic orifice; but also by the patescence of the mitral valve itself, and the consequent regurgitation into the auricle. The second sound was not accompanied with murmur, because the valve expanded widely from the auricular side; and the sound itself was unusually loud, because the recoil of the semilunar valves was impetuous. The regurgitation, together with the aortic contraction, accounted for the smallness and weakness of the pulse; and the retardation of the blood, thus occasioned, led to the dilatation of the left auricle, and eventually to that of the right cavities. The increased action of the right ventricle, conspiring with the obstruction on the left side, occasioned the engorgement and apoplexy of the lungs. The drowsiness terminating in coma, is to be attributed to venous congestion, of which the sudden infiltration of the face was an indication. This congestion was probably increased by the extreme engorgement of the lungs; and its fatal consequences display the formidable nature of a complication which peculiarly favours such congestion: viz. increased power on the right side of the heart, and an obstruction on the left.

Inflammation on the internal membrane of the heart and aorta, occasioned by the frequent rheumatic fevers, was the cause of the vegetations of the heart, and the puckering and contraction of the aorta.

Hypertrophy and dilatation; adhesion of the pericardium; contraction of the mitral and aortic valves, with regurgitation through both. Hemiplegia. Previous endopericarditis.

Benjamin Payne, æt. 37, a basket-maker, of pale, leucophlegmatic complexion, was admitted into St. George's Hospital, under Dr. Hewett, October 8th, 1829, with dyspnœa and palpitation on every exertion, and occasioned in the night by lying in an uneasy position; cough; puffy swelling of the face; no œdema pedum at present, but is subject to it; sense of constriction across the epigastrium; pulse rather small and weak, slightly vibrating, regular now, but it sometimes intermits every alternate beat; urine free.

For many years slightly short-winded on ascending. Fourteen months before admission had hemiplegia of the left side, which, though cured, left his present symptoms.

Auscultation.—*Resonance* deficient in the præcordial region, which is unnaturally prominent. *Impulse* is of a curbed or struggling nature, and is felt in epigastrio. It is an occasional shock with little heaving, and its force in general scarcely exceeds the natural standard; but occasionally it has a vigour considerably greater, and accompanied with a back-stroke. *Sounds.* A prolonged bellows-murmur accompanies both, and the two are continued into each other. The flapping of the second is more audible on the second or third ribs than lower down. The impulse and first sound are synchronous. Above the clavicles there is a hoarse, but subdued and remote sound, and a very slight pulsation.

Diagnosis.—*Moderate hypertrophy and dilatation of the heart. Disease of the valves.* His symptoms were much mitigated by the usual remedies, particularly by occasional small bleedings: but they continually recurred in an aggravated form, and he sank December 19.

Autopsy.—Adhesion of the pericardium. Left ventricle nearly an inch thick, and its cavity dilated to one-half larger than natural. The right ventricle slightly hypertrophous, and its cavity enlarged, but not to the same extent as on the opposite side. The *mitral valve* converted, by cartilaginous thickening, into a rugged, knotty ring, not more than half the natural size. *Aortic valves,* likewise thickened by knotty cartilage. Corpora sesamoidea, enlarged to the size of small peas, considerably obstruct the aperture. Interior of the aorta is slightly steatomatous, but smooth. *Lungs* do not collapse, and are of immense size from sero-sanguineous engorgement. Some fluid in the cavities of the pleura.

Remarks.—The enlargement of the heart was indicated by the prominence and dull resonance of the præcordial region, and by the pulsation reaching to the epigastrium. The hypertrophy was denoted by the occasional vigour of the shock, and by the back-stroke. The irregularity of the heart's action was attributable to the valvular disease. Although the *struggling* nature of the impulse was very characteristic of adhesion of the pericardium, the idea was discountenanced by the history, which, according to the patient's account of it, did not supply evidence of antecedent pericarditis. Co-existent endocarditis was the cause of the valvular disease. It was indicated by the murmur of both sounds.—That of the first was occasioned not only by the state of the aortic valves, but also by regurgitation through the mitral. The murmur accompanying the second sound, resulted from aortic regurgitation. The flapping of the second sound at the second and third ribs, proceeded from the semilunar valves. The smallness, weakness, and intermission of the pulse proceeded from the mitral regurgitation, and its vibration, from the aortic regurgitation. The jerk which properly characterises the latter, was neutralized by the want of a fulcrum in the mitral valve. This was a case of cardiac asthma.

Dilatation and ramollissement of the heart; great contraction of the tricuspid, and still more of the mitral valve, with regurgitation through each; no murmur with the second sound; hydropericardium.

Christian Anderson, æt. 42, in the Edinburgh Royal Infirmary, June 16th, 1825. Cheeks, nose, and lips purple; turgescence and undulation of the jugulars; dyspnœa, occasionally in paroxysms induced by cough or any exertion; starting from sleep, and frightful dreams; œdema of the face and legs; pulse imperceptible; urine scanty, and high.

Eighteen months before admission, she "strained herself opposite to the navel," by carrying heavy weights: hæmoptysis ensued and lasted three weeks, attended with palpitation, dyspnœa, and cough.

Auscultation.—Impulse an irregular succussion or undulation of the chest. *Sounds.* The first (at the lower extremity of the sternum) was a very loud filing-murmur, or that of obscured and subdued sawing. It commenced abruptly, with a flap. The second sound, short and flat, was so weak as scarcely to be audible. It concluded the first murmur. The same sounds existed on both sides of the heart, but were more subdued and indistinct on the left. They were more or less audible over the whole anterior surface of the chest.

Diagnosis.—Much disease of the valves; dilatation of the heart, particularly on the right side; parietes flaccid; not thickened.

Autopsy.—The heart was nearly twice its natural size. ***Right auricle and ventricle*** much dilated: the latter larger than an orange. Parietes of both of natural thickness, but the ventricular columnæ carneæ enlarged. Muscular substance firm but pale. ***Left ventricle.*** Its cavity enlarged to the size of a goose's egg. Walls of natural thickness, but pale, flaccid, and easily lacerable. ***Left auricle*** slightly thickened and dilated. ***Tricuspid valve*** an uneven thick cartilaginous ring, which admitted the middle finger. ***Mitral valve*** was a similar ring, as thick as a crow-quill, admitting the end of the little finger. ***Pulmonic and aortic*** valves were natural, except that the corpora sesamoidea of the latter were enlarged and cartilaginous, but not so as to prevent the valve from discharging its function. The pulmonary artery was somewhat dilated. ***Pericardium*** contained ℥vij of serum; and the cavities of the pleura about Ov or vi. ***Lungs*** œdematous, and slightly tuberculous.

Remarks.—To the original notes of this important and instructive case—the first in which regurgitation was ever noticed, Laennec, Bertin, Bouillaud and all other authors having overlooked it—is annexed the following remark:—"*As the pulmonic and aortic valves were equal to the discharge of their function, the (filing) sound proceeded from regurgitation through the auricular valves. Hence, if 'bruissement' be heard during the ventricular contraction, we are not necessarily to infer, that there is disease of the aortic or pulmonic, rather than of the auricular valves.*" It might be objected to this argument, that the enlarged corpora sesamoidea of the aortic valves were capable of occasioning the murmur of the first sound. To this we may reply in the negative; as the current of blood through the aortic valves was too feeble to excite a murmur, since it was incapable of creating a perceptible pulse.

The greater weakness of the murmur on the left side, appears to me attributable to two circumstances:—1st. The smallness of the mitral aperture; in consequence of which the quantity of fluid retropelled, was inconsiderable. 2nd. The ramollissement of the left ventricle: whence the retropulsion of the fluid was languid. The deficient supply of blood, the mitral regurgitation, and the inadequate power of the ventricle, account for the imperceptible pulse. On the right side of the heart, the ventricle was stronger, and the aperture of the tricuspid valve was double the size. Hence, the murmur was louder.

The second sound was scarcely audible. This is what we should expect: for the scanty supply of blood in the aorta would not close the semilunar valves with sufficient force and velocity to occasion much sound. Nor was this second sound accompanied with murmur; a fact which, in the first edition of this work, I ascribed to the circumstance that, as the ventricles, in consequence of their dilatation and ramollissement, possessed little resilient power, the blood, deprived of their suction, passed indolently through the valves. But I have subsequently met with numerous cases, including the two next, in which the murmur was deficient, though the ventricles were healthy; whence I am led to the conclusion, contrary to Laennec and all other writers, that the diastolic current is naturally too weak to occasion much, or sometimes any murmur, when the auricular orifices are contracted (see p. 78). Laennec's error originated in his mistaking the murmur of aortic regurgitation, which I have shown (p. 75) to be exceedingly common, for that of mitral contraction.

It was reflection on this case that led me to doubt the inferential explanation of the second sound which I broached in the first edition of this work, and that gave origin to the researches (p. 25) which issued in the experiments (p. 32,) demonstrating the real source of the second sound to be the semilunar valves.

The undulating motion of the heart was occasioned by hydropericardium.

The next case also proves mitral regurgitation and murmur, and the absence of murmur with the second sound.

Mitral regurgitation with murmur; but no murmur with the second sound.

Elizabeth Dennis, æt. about 50. Emaciated, admitted into the St. George's Infirmary, under Sir James Clarke, December

8th, 1830.* Affected with all the symptoms of organic disease of the heart in their most severe form. Has been affected with ascites and anasarca. Bellows-murmur accompanying the first sound below the middle of the heart, but not in the region of the aortic valves. Impulse strong; pulse irregular, unequal and extremely feeble, later than the ventricular systole.

Diagnosis.—Hypertrophy and dilatation. If there is no disease of the aortic valves, the bellows-murmur is from regurgitation through the mitral. Is it a ring?

Autopsy.—(Performed in the presence of Sir J. Clarke, Mr. Howship, Mr. Syme, house-surgeon to the Infirmary, and the writer.) Hypertrophy and dilatation of the heart. All the valves healthy except the mitral, the free margin of which was thickened by fibro-cartilage, and the chordæ tendineæ were shortened in such a manner as not to allow the layers of the valve to come in apposition: hence a space, judged to be about as large as a finger, was left, through which regurgitation could take place.

Remarks.—This case affords evidence, which will, I conceive, be considered unequivocal, that regurgitation through an auriculo-ventricular valve occasions murmur with the first sound; also, that it produces a feeble, irregular, and unequal pulse. The next case proves the same.

Aortic valves rigid; mitral, extremely cartilaginous and ossified, with regurgitation and murmur, but no murmur with the second sound; tricuspid cartilaginous; great dilatation.

George Sharpe, æt. 33, sallow, with livid palpebræ, was admitted into St. Bartholomew's Hospital, under Dr. Latham, June 7, 1826. Symptoms were, great palpitaton and dyspnœa, sometimes occurring spontaneously; great œdema pedum; congestion and undulation of the jugulars; somnolency; pulse 130, weak, irregular, and intermittent. Urine scanty and high.

Short-winded, so that he could not run up stairs, for eight or ten years. For three or four years has had a constant short cough, with great proclivity to bronchitis. Has been much worse since a severe cold contracted six months ago.

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* Sir James Clarke kindly invited me to see this case. I wrote the physical signs with the diagnosis in his journal, from which I now transcribe them.

Auscultation.—Resonance of the præcordial region extensively dull. *Impulse,* though feeble, is felt from the fourth to the eighth rib. Below the left nipple, the shock is somewhat stronger than natural. *Sounds.* The first is a grating combined with a whizzing murmur, which, over the left ventricle, is loud and near to the ear; while, over the right, it is as if remote. In the latter situation the flapping of both sounds is remarkably loud. The second sound, on the left side, is without murmur.

Diagnosis.—Dilatation and hypertrophy of the left ventricle, but walls not appreciably thickened. Right ventricle and auricle dilated, but not hypertrophous. Valvular disease on the left side. On the right side also? (If the event disprove this, does the murmur heard on the right side proceed from the left?)

Autopsy.—℥ij or iij of serum in the pericardium; Oij in the chest, and as much in the abdomen. *Heart* enlarged to nearly double. Right ventricle would contain a large lemon; its walls were less than one-fourth of an inch thick, but the columnæ were enlarged. Auricular orifice considerably widened. Loose margin of the *tricuspid valve* cartilaginous and thickened, but it was judged capable of closing the aperture. Left ventricle would contain a small lemon; walls half an inch thick at the base, and a quarter at the apex. *Aortic valves* very rigid with cartilage. *Mitral valve* extremely diseased. The base and margin were of fibro-cartilage, intermixed with denuded bone. A lamellated polypus of organized lymph, as large as a walnut, grew in the auricle by vascular connexion with the lining membrane, which was rough, opake, and yellow.

The internal coat of the arteries was stained of an intense red.

Remarks.—The extensive dulness, the languid impulse, and the loud flapping sound of the ventricular contraction, denoted the dilatation; while some degree of power in the shock below the left nipple, indicated that the walls of the ventricle were not attenuated. The valvular disease on the left side was denoted by the murmur. The compound nature of the murmur, partly whizzing and partly grating, indicated that both valves were affected. I have frequently met with this compound species of murmur, the whizzing character appertaining to the aortic valves, in consequence of their being nearer the surface. This is well exemplified in another individual at present under my notice, affected with disease of both valves, in whom there are from two to five beats of the heart accompanied with grating murmur, but no pulse in the radials: then succeeds a stronger shock with a pulse, and a hissing opposite to the aortic valves.* In the present case, the grating sound, the feebleness and instability of the pulse, and the general symptoms of obstruction on the left side of the heart, left little doubt that there was regurgitation through the mitral valve.

Why was not the second sound, or that synchronous with the left ventricular diastole, accompanied with murmur from the contracted mitral? Because the diastole is not usually attended with a sufficiently copious and rapid passage of blood to occasion a murmur.

The following case shows that contraction of the mitral valve, when extreme, and attended with attenuation and softening of the left ventricle, may not be attended with murmur of either sound.

Dilatation and softening of all the cavities: hypertrophy of the right ventricle; attenuation of the left; great contraction of the mitral valve; fatal polypus.

Mrs. —l—n consulted me Dec. 27, 1829. She had livid lips; a defined purplish red on the cheeks; complexion elsewhere sallow; dyspnœa and palpitation, excited even by walking across a room, and to excess by ascending a flight of stairs; frequent cough, preventing sleep; constant copious expectoration of frothy, viscous mucus, the temporary suppression of which, by sleep or opiates, caused paroxysms of excessive dyspnœa and orthopnœa; chilliness, particularly of the extremities; universal and extreme anasarca; catamenia regular; bowels open; pulse small, weak, unequal, and intermittent; urine scanty and high; thirst; anorexia.

Complaint commenced ten years before I saw her, and was attributed to difficult parturition. The symptoms were always greatly aggravated by colds, to which she was particularly liable. She had frequently had slight œdema pedum, which subsided spontaneously. Always felt best in a warm, humid atmosphere.

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* M. Bouillaud states that he was the first who noticed this variety of intermission. He is mistaken, as the present case is long anterior in date to his publication in 1835.

Auscultation.—Impulse imperceptible. *Sounds.* Both were short, flat, and audible as far as the right clavicle. They were weaker on the left side of the heart. Murmur was not noticed. By the usual diuretics and aperients, the dropsy was completely removed in six weeks, the strength being little impaired and the appetite good. She was then suddenly seized with oppressed palpitation, suffocative orthopnœa, constant nausea, and overpowering exhaustion, anxiety and jactitation. The dropsy began to re-accumulate, the sense of suffocation became agonizing, the pulse failed entirely for twenty-four hours before death, and she sank a week after the relapse.

Autopsy.—Pulmonary apoplexy and engorgement. *Heart* double the natural size, and very flaccid and pale. *Ventricles.* Right dilated to double; its parietes were not attenuated, and the columnæ carneæ were hypertrophous. The left was less dilated, and its walls were reduced to one-third of an inch in thickness. *Auricles.* Right, dilated; its parietes thin and diaphanous. Left, greatly dilated, considerably thickened, and almost completely filled with a polypus adhering firmly to its lining membrane. *Valves.* Aortic, slightly cartilaginous, but unimpeded. *Mitral,* contracted by cartilage into a *slit* which only admitted a writing quill. ℥v of serum in the pericardium. *Liver* slightly enlarged, granular, and of yellowish brown colour.

Remarks.—This case is remarkable as presenting a degree of valvular contraction seldom if ever exceeded, and as showing with how great an amount of disease life may be prolonged for a series of years.

The dilatation was manifest from the deficient impulse, and the short, flat sounds. Though no murmur was noticed on the left side of the heart, contraction of the mitral valve was inferred from the small, weak, unequal, and intermittent pulse, and from the languid action of the left ventricle: as, however, I have since ascertained that a similar pulse and impulse, together with venous retardation, may be occasioned by softening independent of valvular disease, the latter must not be confidently inferred unless there is a murmur.

The reason why great contraction of the mitral, such as existed in the present case, should not always produce murmur, is explained p. 77.

The columnæ carneæ of the right ventricle were hypertrophous. This, concurring with the obstruction of the mitral, accounted for the pulmonary congestion and apoplexy. Hence, too, the copious expectoration; which being the mode that nature adopts to unload the vessels of the lungs, it is obvious why the symptoms were aggravated when the expectoration was suppressed, whether by opiates, catarrh, or a dry, sharp air. The relapse occurred at that critical moment when the dropsy had disappeared: the sudden supervention of suffocative dyspnœa, &c. renders it probable that the polypus in the left auricle commenced at that time, and was the cause of the symptoms and of the fatal event. Hence the importance in such cases of preventing nausea, syncope, or any affection which can cause stagnation of the blood. (See Polypus).

The following case presents a beautiful instance of

Recent lymph or vegetations on the tricuspid valve, from acute endocarditis.

Ann Fenn, a patient admitted into St. George's Hospital, in April 1839, was represented to me by students who took notes of the case, to have exhibited the symptoms of acute endopericarditis, and to have died during the acute stage. I did not see her during life, but witnessed the post-mortem examination.

Autopsy. The *pericardium* contained several ounces of serum and thin patches of soft, yellow lymph adhered to it in several parts. The *mitral valve* was opake and greatly thickened, the chordæ tendineæ thickened and contracted, and the orifice only admitted the thumb. This state was from fibrous hypertrophy, and of a date anterior to the last acute and fatal attack; though the redness of the lining membrane over the whole interior of the ventricle and on the valves, evinced that it had participated in the recent acute endocarditis. The *tricuspid valve* was the object of greatest interest. It was overspread with thick lumps of recent, pasty, yellow lymph, which matted together the serrations of its margins and the chordæ tendineæ, so as to contract the aperture to the size of a finger. The interior of the ventricle and the surface of the valves were universally red, from the acute inflammation.

Remarks. This case presents the most complete instance that I have ever witnessed of a deposition of perfectly recent lymph leading to great disease of the valve, and it is the more remarkable as having occurred on the right side of the heart, where valvular disease is comparatively rare. Nature is, as it were, surprised in the midst of her process. It is clear that, if the agglutinating process can proceed to such an extent in so short a time, the treatment for endocarditis cannot be too prompt and decided. The rapidity with which organization of lymph takes place, is well known; and if temporizing or inefficient treatment leave time for this process, the mischief is irreparable.

The following is an instance of rheumatic endopericarditis, in which the treatment failed to arrest the inflammation in sufficient time to prevent incurable valvular disease.

Acute Endopericarditis; contraction of the aortic valves and regurgitation, each occasioning a murmur; adhesion of the pericardium.

William Harrison, æt. 22, at St. George's, August 11, 1830. Had extremely acute rheumatism with pain in the cardiac region, violent palpitation, and a strong *jerking* pulse of 110. Was repeatedly bled and took calomel and opium with temporary relief; but the pain in the heart became very intense, lancinating to the back, and being increased by inspiration: the pulse became faltering, and the anxiety and distress excessive. In this state he was relieved by a blister and the supervention of ptyalism. A fortnight after admission, the pulse was *extremely jerking*, but regular; the *impulse* of the heart was a violent smart, bounding blow, strongest at the left mamma: the *first sound* was a prolonged but not very loud bellows-murmur. The *second* was like a sigh made with the lips nearly closed. A month after this time, the impulse was *struggling* and strong, but not lower down than natural, though the heart was enlarged. The bellows-sounds as before, but the first louder. Pulse 100 extremely jerking.

Remarks —This was a well-characterized case of acute endo-pericarditis. Much liquid effusion was indicated by the supervention of a faltering pulse, with excessive anxiety and distress. After the absorption of the liquid, contraction and permanent patency of the aortic valves were indicated by the double murmur, and the latter by the extremely jerking pulse. Adhesion of the pericardium took place, and was indicated by the strong and *struggling* impulse.

The following case exhibits the same disease as the preceding, five years after its formation.

Adhesion of the pericardium; hypertrophy with dilatation; disease of the aortic valves with obstruction and also regurgitation, and a murmur from each; contraction of the aorta, with roughness; anæmia; the cause, rheumatic endo-pericarditis.

Joseph May, æt. 20, at St. George's, under Dr. Hewett, September 2, 1831, green-grocer, and goes about much with heavy loads. Complexion leuco-phlegmatic from much puffy infiltration. Violent action of the heart, visible over the whole anterior chest, with a sense of universal throbbing, especially in the temples and vertex: action irregular: sometimes three or four unusually violent beats, occasioning vertigo and stupefaction, which caused him to sink down in a state of unconsciousness for a few seconds. Dyspnœa, greatly exasperated by any effort; until within a month, it occurred, with palpitation, in a violent paroxysm every night, compelling him to rise, and lasting twenty minutes. It was always accompanied by pain in the region of the liver. An ounce of gin, which extricated flatus by eructation, never failed to relieve both the pain, the palpitation, and the dyspnœa. The attack invariably ended in a drenching perspiration and a lax dejection, followed by sleep. Had been subject to it nightly for upwards of four years, the time of its supervention being, at first, eight o'clock p. m. and becoming gradually later till it arrived at two o'clock a. m. Frightful dreams; universal dropsy; urine scanty and high coloured; pulse rather large, extremely jerking and sharp, incompressible, irregular and intermittent.

Five years ago, had two or three attacks of acute rheumatism at intervals of two or three months, which left pain and palpitation of the heart. Six weeks ago, nine quarts of serum were drawn off by punctures, with great relief.

Auscultation.—Impulse is *double*, forwards and backwards, with the first and the second sounds respectively, which occasions a tumultuous *jogging* motion—strongest at the left mamma.

Sounds.—Both have a prolonged filing-murmur, almost continuous, and loudest over the left ventricle, the first being the more hissing. Over the right ventricle the murmurs seem remote, while the flapping of the second sound is loud.

Above the clavicles, especially the right, strong impulse, tremor, and a loud, hoarse murmur. The latter is heard of a more hissing, superficial nature along the sternum in the tract of the aorta.

Diagnosis.—Hypertrophy with dilatation of the heart, the former predominating in the left ventricle, and the latter in the right: disease of the valves on the left side; and of the interior of the aorta, with dilatation. Adhesion of the pericardium.

After being greatly benefited by the judicious treatment of Dr. Hewett, he was seized with erythema of the leg, from the excitement of which he sank, with stupor, in four days.

Autopsy.—Heart had forced the left lung upwards to between the fourth and fifth rib, and five or six bands, half an inch long, united the pericardium to the costal pleura.

Pericardium adhered universally and closely to the heart. *Left ventricle:* walls, an inch thick: cavity, size of an ordinary orange. *Right ventricle:* not thickened; dilated to double; columnæ carneæ enlarged. *Auricles* natural. *Mitral valve* thickened and opake, but not contracted or patescent.

Aortic valves.—On one was a calcareous concretion as large as a small pea, projecting conically into the centre of the artery: on another was a similar but very small deposition. The two aortic valves on the left were thickened and opake, but free.

Aorta was contracted and puckered by steatoma opposite to the left carotid, where its circumference was only two inches and a half, decreasing beyond that point.

Remarks.—The jogging action and the history of previous pericarditis indicated adhesion of the pericardium, and this caused the heart to beat higher than is usual when it is greatly enlarged. The increased impulse and extent of its range, indicated hypertrophy with dilatation. The osseous concretions on the aortic valves occasioned the loud hissing of the first sound: the prolonged murmur of the second proceeded from aortic regurgitation, which was also indicated by the extremely jerking pulse. The murmur and tremor above the clavicles was occasioned by the steatomatous and puckered condition of the aorta; by the unfilled state of the arteries, resulting not only from the aortic regurgitation, but also from marked anæmia; and by the force and velocity with which the blood was propelled, as denoted by the extremely jerking pulse. These phenomena are fully explained in the section on purring tremor, p. 125. The impulse above the right clavicle was occasioned by the same causes, the effect of which was probably aided by the contraction of the aorta immediately beyond. This unusual combination of circumstances led me to suspect dilatation of the aorta, which did not exist; but the roughness of the vessel was correctly inferred from the loud, hoarse murmur above the clavicles, inorganic murmurs never being loud and hoarse.

The regularity of the nightly paroxysms of asthma, the good effects of gin, the termination of each attack by perspiration and purging, the enormous discharge of serum by punctures, and the fatal consequences of a slight inflammatory affection, are worthy of remark. I could not learn that the periodicity of the attacks was connected with malaria.

In the following case, the same organic lesions as in the preceding, and also resulting from endopericarditis, were further attended with aneurism of the aorta

Endopericarditis: aneurism of the aorta causing depression of two aortic valves and regurgitation: mitral regurgitation · adhesion of the pericardium: hypertrophy with dilatation.

Charles Williams, æt. about 25, in St. George's Hospital, Oct. 23, 1834; butcher, formerly so strong that he could carry 48 stone (672 pounds) of meat fifty yards, and never hesitated to lift and carry anything less. About four years and a half before his admission into the hospital, he had been affected with a "violent inflammation of the chest," (Endopericarditis?) subsequent to which he occasionally experienced great "throbbing of the heart," headache, and embarrassed breathing on making any exertion. (Valvular disease, &c. from the endopericarditis?) About nine months before his admission, while digging hard clay land, he speedily became affected with sickness, vomiting of frothy phlegm, and a little shortness of breath, which symptoms increased through the whole day. Next day, thirst; lifted a heavy man up stairs, which was immediately followed by a flush succeeded by cold perspiration, debility, and "nervousness." Went to bed. Next day, did not work. On the following day, before rising, was seized with vomiting, purging, and confusion of head. He was now visited by Mr. Cottingham of Bexley, who subsequently sent the man to me, and to whom I am indebted for the following particulars. "I found him in a state of suffering, which I thought would soon terminate his existence. He lay recumbent; slightly delirious; countenance pallid, exhibiting great anxiety; skin rather cold; respiration laborious; some cough, and frothy expectoration slightly tinged with blood: complained of violent throbbing pain in the head, and of excruciating pain in the regions of the clavicle, scapula, and humerus: his pulse was very irregular, being sometimes full, accelerated, and intermittent in the greatest degree; and at other times, (perhaps after the lapse of a few minutes only,) it was small, almost indistinct, and intermittent. The impulse of the heart was excessively strong, and perceptible over two thirds of the chest. A very distinct, loud bellows-sound was audible over the region of the left ventricle." (Recurrence of endopericarditis?)

"Blood was now taken from the arm, which gave temporary relief; and, in the course of six weeks, about two hundred ounces were drawn. It was not very tough in its texture, but exhibited the thickest coat of buff that I ever saw. The crassamentum at first predominated over the serum; was cupped: after 150 ounces had been drawn, the serum appeared in excess, and, of course, our depletion was diminished. His diet was solely vegetable, and small in quantity. The bowels were kept freely open. He took tartar emetic, digitalis, tobacco, colchicum, and his chest was anointed with mercury and iodine. He got daily surprisingly better."

About seven months subsequent to this attack, he felt worse; Mr. Cottingham sent him for my opinion, and I procured his admission into St. George's Hospital.

I found him to present, in a severe form, all the ordinary general symptoms of organic disease of the heart, which need not be detailed. The physical signs were as follows.

Prominence, in a slight degree, of the præcordial region. *Dulness* on percussion extensive, (3¼ inches in diameter,) yet not preternaturally low down. (Heart bound up by adhesion of the pericardium?)

Impulse is felt during the diastole, i. e. with the second sound, and is strong and jogging. (This caused some to mistake it for the systolic impulse, which was the weaker of the two). The systolic impulse is attended with retraction of the costal interspace next below the nipple, as if the apex were bound down by adhesion of the pericardium, and thus prevented from tilting outwards.

Sounds.—A brief sawing murmur supersedes the first sound over the semilunar valves, and is louder and more superficial there than half way down the heart (aortic obstruction.) The second sound over the semilunar valves is a loud flap, drawn out into a prolonged sawing murmur, which is very loud and superficial, and continues on ascending the aorta, but becomes rather less loud and superficial, as if more remote. It also becomes less superficial on descending down the tract of the left ventricle, and, near the apex, is feeble and remote (aortic regurgitation). Near the apex also, is a murmur which supersedes the first sound: it is distinct, long, and predominant, and decreases on ascending up the tract of the left ventricle (mitral regurgitation).

Purring tremor above the sternal ends of both clavicles: also, a hoarse, abrupt, rasping sound (disease of the arch of the aorta).

Pulse 90, a *jerk*, with the slightest vibration, and very compressible (aortic regurgitation). It intermits occasionally.

Diagnosis.—Roughness of the aortic valves or ascending aorta: regurgitation out of the aorta, through the valves or an aneurism, into the left ventricle: mitral regurgitation: adhesion of the pericardium: hypertrophy with dilatation.

The patient died in January 1839, and Mr. Cottingham, to whom I transmitted my diagnosis of aortic regurgitation and aneurism, &c. made an examination, and obligingly sent me the preparation.

Autopsy.—*Pericardium* perfectly adherent throughout its entire surface to an enormously enlarged heart, equalling in size that of a small ox. *Ventricles.* The walls of the right were about half an inch thick, and the cavity as large as a turkey-egg. The walls of the left were about three quarters of an inch thick, and the cavity equal to the largest orange. *Valves.* The right were healthy, but strong. The layers of the *mitral* were thickened, opake, and rather contracted, but the aperture admitted three fingers. The columnæ carneæ were pointed, as if from being drawn out, in consequence of the immense size of the ventricle rendering them too short to close the valve—a state tantamount in its effect to shortening of the chordæ tendineæ, regurgitation being in both cases the result. *Aortic valves and aneurism.* These are delineated in fig. 13. An aneurism as large as a bantam's egg (*a*) was situated immediately above the junction of two valves, which it had depressed, and caused fibro-cartilaginous thickening and evertion of their edges (*b* and *c*); by which, and the contracted state of the third valve (*d*), free regurgitation was permitted. Steatomatous disease (*e e*) surrounded the aneurism. The arch of the aorta was not examined.

Remarks.—The diagnosis of this complex case was verified in every particular with a precision, which is unattainable except by the aid of auscultation. There was no certain evidence of the aneurism, but I conjectured it from the circumstance that the patient had twice become faint and sick during great muscular exertion (see p. 198-9): viz. first, when digging, and, on the following day, when lifting a person up stairs. It was probably at this time that the aorta, perhaps diseased by the inflammation three years previously, burst and gave origin at once to the aneurism and the second attack of endopericarditis.

The several murmurs verify the rules which have been offered in this work for the detection of the respective valvular diseases.

The jogging impulse, the highly seated dulness on percussion, and the prominence of the præcordial region, verify the rules given at p. 194 for the detection of adhesion of the pericardium.

The next case beautifully exemplifies the prolongation of an aortic regurgitant murmur through intermissions of the heart's beats.

Aortic regurgitation; its murmur prolonged through intermissions of the heart's beats; hypertrophy with dilatation.

W——., Esq., æt. 60, consulted me on March 1, 1838. Palpitation on ascending; often vertigo, especially when the stomach is empty; occasionally headache, especially in the right occipital region,—but not so bad as before an attack of hemiplegia two years ago. Occasionally, pain in the heart, running down the inside of the left arm. Left leg and arm rather colder and weaker than natural, with diminished sensation. The coldness seems to increase on taking exercise, though the body in general be heated. Left pulse rather weaker. Slightly trails the leg, but can walk several miles. Intellect, he says, not impaired; bowels regular; urine free, but nitre with cream of tartar and sugar āā ℈i o. n. cause copious diuresis, and relieve a dryness of the tongue, which he calls fever. *Pulse very large and strong, and slightly jerking.* Digestion and general health good. Has taken *much* exercise up to the present time, his physician not having interdicted it. The paralysis two years ago followed much walking up hill in Clifton. *Percussion.* Dulness over a diameter of three inches, and seated low down. *Impulse* strong and heaving, with diastolic impulse. *Sounds.* First, dull over the left ventricle. Second, was a soft and very prolonged murmur, on the key of *awe*, whispered by inspiration; it was very audible not only up the course of the aorta, (though not of the pulmonary artery,) but down the middle of the left ventricle; and, *when the heart intermitted, the murmur continued beautifully during the whole of the intermission.*

Diagnosis.—Aortic regurgitation, but not very considerable, as the pulse is only slightly jerking: hypertrophy with dilatation.

Remarks.—The continuance of a murmur from aortic regurgitation through the whole period of an intermission of the heart's beat, is a circumstance sufficient of itself to convince the most sceptical, were other evidence wanting, that the murmur could proceed from no other source than a regurgitation out of the aorta or pulmonary artery.

The next four cases are excellent exemplifications of musical murmurs, and illustrate the general rules offered at p. 87-8.

Aortic regurgitation with loud musical murmur: mitral regurgitation and murmur: hypertrophy with dilatation.

Henry Milton, æt. 28, was admitted into St. George's Hospital, March 15, 1837. Is a carpenter: tongue slightly furred; bowels costive; palpitation; pulse full and *jerking*. Six years ago, had an attack of acute rheumatism of eleven weeks duration, for which he had medical treatment. Two years subsequently, had another attack. Short-winded ever since. Fourteen months ago, first heard a peculiar noise in the chest; consulted Dr. M'Cabe of Hastings, who pointed out to him the palpitation, of which he was himself previously unconscious. March 9th, 1836, went into the Brighton Hospital. Was salivated, and says he caught cold and had another attack of acute rheumatism: was in the hospital four months. Three weeks ago, went into St. Bartholomew's Hospital, under Dr. Latham; and, as he gave him no prospect of relief, he came into St. George's.

Sounds.—A musical murmur with the second sound, loudest over the semilunar valves, and thence up the aorta; while a feeble sighing-murmur may be heard accompanying and prolonging the musical note down the ventricles, but not above the valves. The musical sound is like the *oo* of *coo*: it swells and rises a semi-tone in the middle, like the mew of a kitten. It is so loud as to be audible a foot from the chest through the air, and also in the palm of the hand when the stethoscope is applied to that part. I have only once heard a louder musical murmur (aortic regurgitation). A murmur attends the first sound below the middle of the left ventricle (mitral regurgitation). *Impulse*, violent, extensive, jogging, with strong diastolic impulse. *Dulness* on percussion, extensive and low down.

Diagnosis.—Aortic and mitral regurgitations. Hypertrophy with dilatation; possibly, adhesion of the pericardium.

He died about three weeks after admission, from purpura hæmorrhagica.

Autopsy.—*Lungs* gorged with blood, and presenting many purpurous extravasations. *Pericardium* adhered rather loosely over about two-thirds of the heart. (The looseness of the adhesion was the reason why the heart was not bound up in a higher situation than natural, a circumstance which created a doubt as to the existence of adhesion). *Left ventricle* rounded, and about an inch thick: its cavity, a very little enlarged. *Right ventricle*, a little dilated. *Aortic valves.* All were yellow and morbidly opake, from fibrous thickening. The corner of one was torn from its origin to the extent of two-and-a-half lines, and the flap hung back and overlapped the ventricular side of the valve, so as to allow free regurgitation. The flap was folded on itself, and the folds were adherent to each other, evincing previous inflammation (see fig. 11 *a*). In the dependent flap was a hole one-and-a-half lines in diameter (see fig. 10, *a*). *Mitral valve* and chordæ had undergone similar opake yellow fibrous thickening, and several clusters of vegetations—one or two as large as a pea, existed on the auricular side, a little below the margin. The valve was contracted so as only to admit two fingers. Hence, the regurgitation. *Tricuspid valve*, a little thickened. *Pulmonic valves*, natural.

Remarks.—The diagnosis was exactly verified. The valvular regurgitations were in accordance with the rules inculcated throughout the work for the detection of the several valvular diseases.

Contraction of the aortic valves and a musical murmur with the first sound: regurgitation through the same valves: mitral regurgitation with a second musical murmur.

V. . . . , æt. 50, consulted me, April 5, 1838, in company with Mr. Eisdell, Surgeon, 77, Sloane-street: of full habit; a publican, and a moderate, temperate liver: has drunk gin in moderation. Subject to gout in the feet for ten years. Has been very active, walking four miles an hour. For three years has been short-winded on ascending: rather drowsy in the mornings, but has no other head symptoms. *Pulse* small, weak, irregular, unequal. *Tongue* whitish.

Impulse natural. *Sounds.* A musical note is heard to be loud and near-sounding an inch below, and a little to the sternal side of the left nipple, and it accompanies the first sound of the heart (mitral regurgitation). It diminishes on ascending up the ventricle, and, half way up, it is almost inaudible. On ascending still higher, a second musical note with the first sound becomes audible, and is perfectly distinct opposite to the aortic valves and thence two inches along the aorta, where it sounds more superficial or near than opposite to the valves themselves. This musical note is mixed with a common murmur on a lower key than a whispered *r*, (apparently from the pulse being weak,) which may also be heard along the aorta. Both the sounds are very indistinct along the course of the pulmonary artery. The second sound over the aortic valves is tailed by a feeble, though distinct whispered *awe* murmur, which diminishes on descending down the left ventricle, and is prolonged to the ensuing ventricular systole.

Diagnosis.—Mitral regurgitation: contraction of the aortic valves and regurgitation: little or no hypertrophy or dilatation.

Remarks.—This is the only case that I have seen or heard of, presenting two musical murmurs. It is proved that there are two, by both being almost inaudible midway between their sources; viz. about the middle of the ventricle. This circumstance shows that a musical sound is best propagated in the direction of the current: for I have found such a sound, when produced by aortic regurgitation, audible down the whole extent of the left ventricle. The pulse has no jerk, partly because the mitral regurgitation renders it too small, weak, and irregular to have a jerk; and partly perhaps, because, if I may judge from the weakness of the murmur, the aortic regurgitation is not considerable. Mr. Eisdell was present at this examination, and verified all the facts.

Pericarditis with effusion; and endocarditis, first with mitral regurgitation and a musical murmur: afterwards with aortic regurgitation: an attrition-murmur on absorption of the fluid: final adhesion of the pericardium: sound of costal percussion: hypertrophy.

Robert Jones, æt. 15, under the writer's care at St. George's Hospital, Nov. 13, 1835. Three months before admission, had acute rheumatism so severely as to be confined to bed for a fortnight.

On admission, there was *dulness* on percussion in the præcordial region over a space of three inches across by five perpendicular—the outline being pyriform, with the smaller end ascending up the sternum to the second rib. *Impulse* increased. *Sounds.* A very loud, rough murmur, with a *broken whistle or creak*, attends the first sound, and is loudest over the apex of the heart (mitral regurgitation). *Purring tremor* is felt. Palpitation; dyspnœa; moderate fever; pulse quick.

Diagnosis.—Chronic endopericarditis, with hydropericardium; mitral regurgitation; hypertrophy.

The remedies employed were, a bleeding of ℥ij only; pil. hydr. till the gums were touched; diuretics; and a succession of blisters on the præcordial region. At the end of a month, the dulness on percussion had descended three or four inches, and there had supervened a confused, *continuous* rumbling murmur, heard equally over the whole heart. The previous broken whistle of the mitral valve had degenerated into a less musical chirp. Less impulse; pulse slower.

Diagnosis.—Most of the fluid absorbed: a little probably churned between layers of rough lymph on the pericardium, and occasioning the continuous rumble.

The same treatment was prosecuted. In nine days more, the continuous, diffuse rumble was weaker, and the mitral chirping sound had ceased and been replaced by a pure, loud sawing-murmur, particularly loud over the apex, but obscure elsewhere. The præcordial region was protruded. Dulness still more extensive than natural.

Diagnosis.—Further absorption of fluid.

In twelve days more, a prolonged sawing-murmur began to attend the second sound over the aortic valves, but not over the pulmonic. *Diagnosis.* Aortic regurgitation.

In another week the diffuse rumble was further diminished, being barely distinguishable from the two regurgitant murmurs.

Diagnosis.—Commencing adhesion of the pericardium.

At the end of the next three weeks, a new phenomenon struck my attention: namely, though the mitral regurgitant murmur was a pure whizz so long as the pulse was under 80, yet, when the action of the heart was accelerated by any effort to 90 or upwards, a very loud and distinct *click* was superadded to the whizz, and it gradually went off again under the ear of the auscultator in proportion as the heart returned to its previous tranquil pulsation.

I noticed that the click *was later than the commencement of the whizz* by a very appreciable interval. This phenomenon was verified by Drs. Macleod, Marshall Hall, and Jefferson, and Messrs. Keate and Peregrine. I at first imagined it to be the natural click of the mitral valve, becoming audible when the action of the heart was violent. But, in this case, it ought to have preceded, not followed, the commencement of the whizz. I subsequently ascertained that it was nothing more than the extrinsic sound of costal percussion, described at p. 41, and of which other illustrative cases will presently be offered.

At the expiration of seven weeks more, making a total period of four months and a half, he was dismissed in a state of good general health, and with a pulse of 78. The continuous rumble had wholly ceased; the two regurgitant murmurs alone were heard; the purring tremor was no longer perceptible; the præcordial region was prominent; the impulse strong, and the dulness on percussion two inches and a half across.

Diagnosis.—Adhesion of the pericardium; aortic and mitral regurgitation; hypertrophy.

I lost sight of him for a year and a half; when Mr. Davis, Surgeon, informed me that he had died of dropsy, and obligingly invited me to inspect the body. We made the following notes.

Autopsy, April 1, 1837.—External dropsy and ascites. *Pleuræ* contained a quart of serum. *Lungs* gorged with blood, œdematous, and rather condensed by the pressure of the fluid and heart. *Heart*, in the pericardium, very large, pushing the left lung as high up as the fourth rib. *Pericardium* universally adherent, except at a small point forming the angle between the base and the great vessels, where, it may be remarked, the rumbling murmur had continued longest. The false membrane forming the medium of adhesion, was of an unhealthy character, being bloody, imperfectly organized, and, as it were, rotten. *Ventricles.* Walls of the left, about seven lines thick: of the right, three lines—constituting hypertrophy in a small, slim youth of æt. 17. *Mitral valve* of opake yellow colour, from fibrous thickening both of the laminæ and the chordæ tendineæ, and contracted, so as to admit the passage of two fingers only (hence the regurgitation). *Aortic valves* presented fibrous thickening with contraction and a fringe of small vegetations (hence the regurgitation). The tricuspid and pulmonic valves were healthy. *Liver* gorged with blood.

Remarks.—This case is interesting and instructive in a triple point of view.

1. It presents an instance of a musical murmur (a broken whistle) degenerating into a loud sawing-murmur, and this was the object of its introduction in the present situation.

2. It is an excellent example of the sound of costal percussion (p. 41), its extrinsic origin being beautifully apparent in consequence of the complete extinction of the natural first sound by the sawing-murmur, and in consequence of its occurring later than the commencement of the murmur.

3. The case presents a graphic exemplification of the progress and phases of a chronic endopericarditis, and of the ease with which the valvular may be discriminated from the pericardiac attrition-murmurs, by the rules developed at p. 174. It might be supposed that the transitions, in such cases, are so fine as only to be appreciable by an adept: yet it is not so. There is a force of conviction to be derived from hearing, which cannot be produced by description or by the strongest asseverations, and I shall presently show, in the case of Rogers, that this conviction may be attained by a novice.

Aortic regurgitation, with murmur ultimately becoming musical: hypertrophy with dilatation.

Joseph Tindall, æt. 30, robust, and used to lift heavy weights; labourer on the railway. Applied to me March 26, 1838. Great palpitation and dyspnœa on any exertion, which has disabled him from work for eleven months. Thinks he had rheumatic fever several years ago. *Pulse* pre-eminently *jerking,*—especially on any slight exertion. It is like a hard ball shot with force under the finger, the artery feeling empty in the interval. From this pulse alone, I guessed aortic regurgitation. *Impulse,* considerably increased. *Dulness* over about three inches in diameter and preternaturally low down. *Sounds.* A whispered *awe* murmur with the second sound is heard over the aortic valves and two inches up the aorta, where its key rises to a whispered *r* tone. The murmur may be traced down the left ventricle, with a gradual diminution of intensity and lowering of its key below the *awe* tone. Near the apex it sounds feeble, remote and like a whispered *who.* It is weak up the pulmonary artery. This was the nature of the murmur for five or six weeks, at the end of which time it became *musical,*—especially when the circulation was accelerated. The musical note was clearest and most free from murmur two inches up the aorta: opposite to the valves, the tone sounded lower and more remote, and was mixed with a murmur: on descending down the ventricle, the musical note became very indistinct while the murmur became louder—which shows that the motion of the blood *within* the ventricle occasioned murmur, while the musical note was probably generated by the edge of the valves.

Diagnosis.—Regurgitation through the aortic valves: hypertrophy with dilatation.

The patient is still living.

Remark.—This case is a perfect exemplification of an ordinary murmur becoming musical, and of the co-existence of the two. It also shows that a certain force of current is requisite for the production of the musical note, as it diminished when the heart became calm, and *vice versâ.*

The five following cases present instances of disease, with murmurs, in the pulmonary artery, and they completely exemplify the physical signs of these rare affections.

Great dilatation of the pulmonary artery. Hypertrophy and dilatation of the heart.

Sarah Wetherly, æt. 36, of yellowish complexion, was admitted into St. George's Hospital, under Dr. Seymour, January 20th, 1830, with dyspnœa; pain at the scrobiculus cordis; ascites, œdema pedum; pulse 70, large, full, and rather tense; tongue clean; urine scanty; catamenia suppressed for five months.

Short-winded for ten years, in consequence of striking her breast against a post. Eight months ago the catamenia were checked by cold, from which time she dates her complaint; but the œdema did not supervene until three months afterwards, when the menstrual flux became totally suppressed.

Auscultation.—Resonance of the præcordial region is extensively dull; prominence, pulsation, and purring tremor between the cartilages of the second and third left ribs. *Impulse*, much more extensive and considerably stronger than natural, particularly in the left præcordial region. The pulsation is felt in epigastrio. *Sounds.*—The first, is an extremely loud, harsh, and *superficial* sawing-murmur. It is extensively audible, but most so on the prominence between the second and third ribs.

Diagnosis.—*Hypertrophy and still more dilatation of the heart, greatest on the left side. Dilatation of the origin of the aorta, probably forming an aneurismal pouch towards the left.* V. S. ad ℥x.—℞ calomel. gr. iij. hâc nocte.—℞ haust. sennæ cum tart. potassæ ʒiij cras mane.—℞ haust. salin. efferv. ter die.

Died a month after admission.

Autopsy.—Heart encroached much, by its size, on the left side of the chest. It was hypertrophous and dilated; most on the left side.

Pulmonary artery remarkably dilated. Its internal circumference near the valves was four inches and a half; and midway between this and the bifurcation, it was five inches. The enlargement did not extend beyond the bifurcation. The sigmoid valves appeared to be put on the stretch, and too small to close the orifice, yet this could not have been the case, as there was no murmur from regurgitation. *Aorta* rather contracted. *Mitral valve* slightly thickened. *Abdomen* contained three or four quarts of straw-coloured fluid. *Liver* rather enlarged and hardened, and its peritoneum thickened by old inflammation.

Remarks.—Part of the diagnosis, in this case, was inaccurate: but as dilatation of the pulmonary artery is one of the rarest affections incident to the human frame, and as its signs had not previously been described by any author; while aneurism of the ascending aorta is an ordinary disease with well-known signs, the former could not, on any certain grounds, have been diagnosticated in preference to the latter. On reviewing the signs of the former, however, they appear to me so pathognomic as to render the affection easy of diagnosis for the future. The particulars are given at p. 452, to which the reader is referred.

As the pulmonary artery is close to the surface, the sound possesses in a peculiar degree the character of proximity to the ear of the auscultator.

The strong impulse, and tense pulse denoted the hypertrophy. The great extent of the pulsation, the præcordial dulness, and the largeness of the pulse, indicated the concomitant dilatation. The left side was supposed to be more enlarged, because the impulse was strongest over it.

The next case is another of dilatation of the pulmonary artery.

Dilatation of the pulmonary artery, with continuous murmur; Hypertrophy with dilatation.

Miss L—— P——, æt. 16, without any signs of puberty, but florid and healthy looking. Bowels regular, tongue clear, appetite good, sleep sound, palpitation on any exertion, especially ascending, or on fright. When lying, has sometimes a bound of the heart, which makes her start up and is followed by faintness. Slight pain down the left arm. Has also a considerable curvature of the spine.

When an infant, was pale and unhealthy-looking, and has always been delicate. Hands occasionally "go dead." No cyanosis.

Auscultation.—Left præcordial region slightly prominent, and a bend in the cartilages of the ribs. *Impulse* increased: its heaving and back-stroke may be seen as well as felt. The impulse is high, and extends towards the middle of the sternum, as if the right ventricle were its seat; but the left ventricle is also affected, as the pulse is rather larger and stronger than natural, and the *carotids throb.*

Sounds.—Between the cartilages of the second and third left rib, an *exceedingly* loud and superficial sawing murmur accompanies the first sound, and confuses the second. It decreases downwards, and, on the body of the ventricles, sounds remote. It follows the ramification of the pulmonary artery to the left, but is almost inaudible above the clavicles (hence not seated in the aorta). The second sound is audible through the murmur. *Purring tremor* in an *intense* degree, and impulse, are felt between the cartilages of the second and third left ribs, but not above the clavicles (hence, not dilatation of the aorta).

Diagnosis.—*Dilatation of the pulmonary artery,* probably congenital: *hypertrophy with dilatation of both ventricles.*

Remarks.—The situation of the murmur between the second and third left ribs restricts it to the pulmonary artery rather than to the aorta, and the impulse and tremor denote dilatation of the vessel itself rather than a mere contraction of the pulmonic valves or orifice. Still, it is impossible to say that the latter also does not exist, as the murmur of the artery absorbs any that might proceed from the valves: it is probable, indeed, that it does exist, because the disease appears to have been congenital, and when this is the case there is, in the great majority, a contraction of the pulmonic orifice and a communication between the two sides of the heart, even though there be no cyanosis. The only point in which this case differs from the preceding, is, in the continuity of the murmur.

I have, in two subsequent cases of supposed dilatation of the pulmonary artery, found the murmur continuous. Not having had the opportunity of autopsy, I cannot speak positively respecting the cause, but I have reason to suspect that it is a *venous* murmur, seated in the vena innominata, and adding its continuous sound to the murmur of the pulmonary artery. It will be asked why a dilatation of the pulmonary artery should create a murmur in the vena innominata; seeing, as may be done by reference to the frontispiece, Fig. 1, that the vein in question, (*k*) is separated from the pulmonary artery (*m*) by the interposed aorta (*l*). There is difficulty in answering this question. The case of Phœbe James, described at p. 115, may perhaps throw some light upon it. In this case, the vena innominata was put upon the stretch, and thus rendered more susceptible of vibration, and of the sonorous effect of accidental indentations on it by parts which it crosses. Can dilatation of the pulmonary artery, by displacing contiguous parts, as the aorta, indirectly act in the same way? Or, after all, is the venous murmur of the innominata without mechanical cause, and an accidental adjunct to the dilatation of the pulmonary artery? I have certainly heard it in anæmic subjects presenting it also in the neck, wholly independent of disease in the pulmonary vessel.

Anæmia; contraction of the pulmonary orifice with murmur and thrill.

Grace Bowden, æt. 16, under the care of the writer at St. George's Hospital, January 30, 1839. Pallor; palpitation; breathlessness; faintness; weariness; throbbing in the head; aching in the back; pulse quick, jerking; tongue clean; anorexia; bowels constipated; catamenia have not appeared; leucorrhæa six months. Drooping for a year.

Auscultation.—Loud venous murmur in the internal jugulars. An exceedingly loud murmur with the first sound in the pulmonary orifice and along the pulmonary artery, but weaker along the aorta: louder down the course of the right ventricle, than of the left. A strong thrill between the cartilages of the second and third left ribs.

Diagnosis.—Anæmia: contraction of the pulmonary orifice. (Confect. Ferri ʒi, t. d. s.—pil. aloes cum myrr. i. vel ij. o. n. Animal food twice a day).

Remarks.—Here, the disease was seated in the pulmonary orifice rather than in the course of the vessel, because the murmur was loudest immediately over the valves, and because there was no impulse between the cartilages of the second and third left ribs. One of the principal objects for the introduction of the present case, was, to show that the sound was transmitted more loudly down the course of the right ventricle, than of the left—the converse of what obtains when the aortic orifice is the seat of contraction. The same will be noticed in the next case (Rogers), who had pulmonic regurgitation as well as contraction. The establishment of this fact was necessary to complete the scheme of valvular diagnosis broached in this work.

It must, further, be noticed that a contraction of the pulmonic orifice, by breaking the stream, suffices to create a thrill between the cartilages of the second and third left ribs, but not impulse.

The general symptoms, including the jerking pulse, were mainly dependent on the anæmia. They greatly subsided with the removal of that condition in six weeks. The venous murmur ceased.

Softening and ossification of the pulmonary artery, with murmur.

Lady R. æt. about 60. I attended this patient in 1833 or 1834, in consultation with Mr. Cottingham of Bexley. Having lost my notes of the case, I shall merely state that she presented the ordinary symptoms of organic disease of the heart, and experienced such a craving for breath as to sleep, even during winter, with her window open. There was a murmur over the semilunar valves.

Mr. Cottingham favoured me with the following particulars of the examination:

Autopsy.—The right auricle and ventricle were much dilated and attenuated. The pulmonary artery, where it crossed the aorta, presented a circumscribed spot as large as a shilling, of a darker hue than the rest of the vessel, and slight friction between the finger and thumb abraded it into a hole. It seemed almost pulpy. Where the pulmonary artery plunged into the lungs, *it was found quite ossified*, as well as the larger bronchial tubes.

Remarks.—This is the only instance that has occurred to me of ossification of the pulmonary artery. Its condition was the cause of the murmur, and probably of the unusual degree of craving for breath experienced by the patient.

Acute endopericarditis; double pulmonic murmur, from contraction and regurgitation; attrition-murmur, suspended by liquid effusion, and re-established on its absorption.

Edmond Rogers, æt. 29, was admitted as an out-patient of St. George's Hospital, under the writer's care, March 20, 1839. Eight years previously, he had been affected with acute rheumatism, and had been short-winded and delicate ever since. Three weeks before admission, had been "taken worse," and affected with a pain in the region of the heart, but during the last week it had been confined to the epigastrium, where it was extensively diffused. At the time of his admission he was pale and emaciated; pulse 80, weak, *not jerking;* bowels constipated.

Impulse natural. *Sounds.*—A loud, superficial, *r* murmur with the first sound, and a more prolonged *awe* murmur with the second, were heard over the pulmonic valves, and were louder along the whole tract of the pulmonary artery and right ventricle than along that of the aorta and left ventricle. The first murmur was propagated more loudly up the pulmonary artery, and the second, down the right ventricle, the focus of each being, the pulmonic valves. A *purring tremor* was felt between the cartilages of the second and third left ribs, with both murmurs, but was stronger with the first.

Being unable positively to determine whether the pain in the epigastric, and previously in the præcordial region, was inflammatory, or merely angina connected with previous organic disease, and aggravated, perhaps, by the constipation, I ordered a purgative, and a belladonna plaster over the region of the heart, and requested him to call again at a short interval. He did not return till the fifth day.

Though the constipation had been removed, the diffuse epigastric pain remained: there was much anxiety of countenance, pallor, weak pulse, suspirious respiration, and increased dulness on percussion of the præcordial region.

Sounds.—An attrition murmur of a croaking character or like tearing calico, with a purring tremor, was now discovered over the whole inferior part of the heart, especially on the left side. This was heard by several gentlemen, some of whom could vouch for its absence at his previous visit. No signs, general or physical, of pleurisy. The existence of acute pericarditis was therefore demonstrated, and I inferred that the pain which had existed for three weeks, and probably the pulmonary murmurs, were also results of inflammation. I induced him to remain in the hospital, and prescribed as follows on the 25th.

℞ Hydr. chlorid gri. vi. opii extr. gr. i. Mft. pil. ter die sumendus—℞ Ung. Hydrarg. fort ℥i, axillæ utrique omni mane et vespere affricandam. Empl. Lyttæ cordis regioni applicetur, et posteà cataplasma assiduè adhibeatur.

As he was cold and exhausted, I postponed blood-letting. He now passed into the hands of the physician for the week.

27th. The mercury was omitted at one o'clock, as the gums were slightly sore; and he was bled to ℥xii: blood, buffed and cupped.

28. I found him much worse. He lay diagonally, inclined to the right side; respirations 80 per minute, with dilatation of the alæ nasi: p. 120, weak and intermittent: increased anxiety: pain less acute, but more diffused over the lower part of the præcordial region: *attrition murmur and tremor gone!* first sound very obscure at the apex: dulness more extensive: pulmonic murmurs continue.

Diagnosis.—Liquid effusion within the pericardium has increased, and the layers of the membrane are separated.

29. Gums sorer: better: viz. respiration freer; less anxiety; p. 112, rather sharp; less pain and oppression in the præcordia; dulness diminished: but still no attrition-murmur, and first sound still weak and remote: pulmonic murmurs as before.

Diagnosis.—Effusion diminishing (in connexion with the full action of mercury).

31. Still better: viz. p. 96, regular, and its sharpness gone; respiration freer; further diminution of the præcordial soreness, and dulness on percussion. The attrition-murmur has returned, and is now very loud, and singularly superficial or near-sounding, over the whole heart. It is double, and the first half, or that corresponding with the pulse, is the louder and longer. It resembles the rubbing together of two dry hands, and is *unequally* rough. It is so loud as to be audible, not only over the whole

anterior chest, but even on the abdomen—an observation which verifies the remarks made at p. 169. The pulmonic murmurs are obscurely audible through it.

Diagnosis.—Liquid effusion gone, and there is attrition of dryish lymph.

April 1. Feels better; p. 100, rather unequal in speed, and there are occasional intermissions; respiration free, but easily accelerated: attrition-murmur is rather diminished, and it has a triple or broken character: the pulmonic murmurs can now be distinctly heard through it.

5. Attrition-murmurs now quite gone; pulmonic loud and distinct. The regurgitant murmur, indeed, has become louder than ever, and the tremor attending it is stronger than that with the direct murmur.

The patient was dismissed in a week or ten days, and said to be convalescent.

Remarks.—A number of practitioners and students carefully watched the several transitions of this case; and even those who had not before witnessed a similar affection, declared the physical phenomena to be so distinct, as to create a conviction in their minds which was irresistible. All could with perfect ease discriminate the pulmonic, from the attrition-murmurs, partly by the rules of situation explained at p. 174, and partly by the total difference in character of the two classes of murmurs. All were equally pleased to observe the beautiful correspondence between the general and the physical signs—the former assuming their worst type (on the 28th) when the cessation of the attrition-murmur, &c. indicated much liquid effusion in the pericardium; while, on the other hand, they improved in the same proportion as the physical signs denoted the gradual absorption of the liquid. Without auscultation, such cases are, and always have been considered, darkness and confusion. The happy effects of mercury, to which the patient owed his life, were strikingly evinced by the remedy coming into full operation, and affording immediate relief, at the critical moment (the 28th) when experience has shown that other remedies are almost unavailing.

The case presents two circumstances of especial interest: 1. a pulmonic regurgitation, which is exceedingly rare; and, 2. a tremor attending that regurgitation.

We now leave the diseases of the pulmonary artery, and pass to the sound of costal percussion and metallic tinnitus described at p. 41. The following cases, in addition to that of Jones, p. 559, are examples of this phenomenon.

Carrington consulted me March 30, 1838; æt. 30, tall, thin, a footman, has hypertrophy, with palpitation and dyspnœa on exertion. There is pretty strong impulse between the fifth and sixth left ribs, where the apex impinges. On placing the stethoscope immediately over this spot, a metallic tinnitus (the cliquetis of Laennec) was heard, exactly like that produced by tapping the back of the hand with a finger while the palm covers the ear. The first sound of the heart seemed to be *double*, like that produced by tapping a table with two fingers at once, but one rather higher than the other: the second of the two sounds was the tinnitus. I have for many years noticed this double sound without tinnitus.

I made the following series of observations on the phenomenon.

1. The tinnitus ceased and the sound was single when either the upper or lower edge of the stethoscope was pressed obliquely into the intercostal space.

2. The tinnitus ceased, but the sound continued double, when the stethoscope with the stopper in was applied flatly over the ribs.

3. I filled the hollow cone with cotton wadding, which, by its elasticity, pressed the intercostal space inwards: when the tinnitus ceased and the sound was single.

4. When I withdrew half the wadding, and left the cone only lightly filled, the double sound and tinnitus returned, though rather diminished.

5. The tinnitus continued, but rather duller, when I placed a penny flat across the two ribs, and listened with the stethoscope upon it.

6. It ceased, as well as the double sound, on full inspiration, and was always strongest during expiration.

7. It was increased by leaning forward during expiration.

Conclusions. The tinnitus cannot proceed from the heart impinging against the chest after having been withdrawn from it, because it was loudest in obs. 7, viz. while leaning forward during expiration—a posture which keeps the heart in constant opposition with the walls of the chest, as may be proved by dulness on percussion.

I infer that the tinnitus and second half of the double first sound proceed from the apex of the heart sliding upwards (as it may be seen to do in the poisoned ass) and impinging against the inferior margin of the fifth rib; for, whenever the intercostal space was pushed in, as in obs. 1 and 3, so as to prevent the edge of the rib from being prominent, the tinnitus and double sound ceased.

I infer that the tinnitus itself proceeds simply from reverberation of sound within the cone of the stethoscope; because it ceased, yet the sound remained double, when the stopper was employed (obs. 2); and because it existed, but with diminished intensity, when the cone was lightly filled with cotton (obs. 4), and also when a penny was interposed between the chest and the open cone (obs. 5)—both of these arrangements admitting of slight reverberation of sound within the cone.

Assuming these explanations to be correct, it would follow that the sound of costal percussion and the attendant tinnitus should be less apt to occur in stout and plethoric subjects; because, in such, the intercostal spaces are fuller, and the edge of the fifth rib therefore less prominent. Now, all the instances in which I have met with tinnitus, since my attention has been turned to the subject, have been in the persons of thin individuals. Thus, the present patient was thin: Robert Jones (p. 559) was emaciated: so also were the subjects of the two following cases, and several others, of whom the notes lie before me.

The next case shows more distinctly than the preceding the constitutional circumstances under which the sound of costal percussion and tinnitus occur.

Sound of costal percussion with tinnitus subsiding on the reduction of anæmia.

A . . . n, Esq., consulted me April 9, 1838. Æt. 30; emaciated; pale; very nervous from youth; violent palpitation, increased by exertion and mental emotion; p. 100 to 120, small and weak; languor; lassitude, &c.

Impulse increased. *Sounds.* Both loud; the first was double, and a tinnitus attended the second half. It was restricted to the space of an inch, where the apex beat. It ceased on pressing the edge of the stethoscope into the intercostal space: also on putting a penny over the spot and listening upon it; but, in the latter case, though the tinnitus ceased, the double sound remained. The same occurred when the stopper was used.

Diagnosis. Anæmic and nervous palpitation. (Sedatives, iron, aloes, and animal diet.)

A fortnight after the preceding report, the anæmic symptoms and nervous excitability had considerably abated, the impulse was less abrupt, the double nature of the first sound had diminished, and the tinnitus had become so much less distinct as not to be very well characterised.

At the expiration of nine months, when the patient, though thin, had recovered from his anæmia and excitability, the first sound was single when the circulation was calm, and it was only during accelerated action of the heart that a slight degree of tinnitus was perceptible.

Remarks.—From this and other cases it appears to be deducible that the diminution of anæmia, by rendering the impulse of the heart less sudden, diminishes the smartness with which its apex glides up against the edge of the fifth rib, and thus diminishes the tinnitus. If this first be found true and general, it follows that anæmia will be one of the elements contributing to the production of tinnitus, palpitation being a second, and meagreness a third.

The following case shows that tinnitus is not confined to the heart.

Tinnitus of the subclavian.

Dr. A . . . . r, æt. 50, exceedingly emaciated; hypertrophy with dilatation; p. 90 to 130, singularly jerking, yet no aortic regurgitation (anæmia). He had most distinct tinnitus produced by the subclavian artery impinging against the clavicle, little cellular or adipose tissue being interposed to intercept the blow.

Remarks.—When we reflect on the slightness of the tap on the back of the hand, which suffices to produce tinnitus when the palm is applied to the ear, we shall easily understand that the blow of an artery against a bone or of the apex against the fifth rib, may be adequate to the production of the phenomenon.

The two following cases are instances of the venous thrill, respecting the existence of which I expressed a doubt at p. 118. I met with the cases while the work was passing through the press, and when it was too late to cancel the passage referred to. I found that I had previously failed to notice the phenomenon in consequence of employing too much pressure. The thrill is so delicate, that anything more than mere contact of the pulp of the finger with the skin, renders it imperceptible.

Venous thrill.

Sarah Pyke, æt. 28; at St. George's, April 10, 1839. Extremely pallid; constipation; catamenia suppressed for four months; all the other symptoms of anæmia in the highest degree.

Messrs. F. Browne and H. Daniels, students of St. George's, verified the venous thrill, and, at my request, made the following notes.

"Loud venous murmur of the internal jugular vein, and a thrill may be felt at its lower part by placing the finger with extreme lightness over the vein. It is most perceptible during inspiration."

Three weeks later, the thrill was gone and the venous murmur diminished, in consequence of the anæmia having been considerably reduced.

Miss N . . . e presented the same thrill under identical circumstances, except that the anæmia was less considerable.

I am at a loss to decide whether the continuous murmur in the following case was arterial or venous.

Disease of the femoral artery and continuous murmur with augmentations.

John Allen, æt. 47, in the St. Mary-le-bone Infirmary, under the care of my former colleague Mr. Perry, had a dilatation of the femoral artery, extending from within two inches of Poupart's ligament to the popliteal region. Along its whole course, there was a strong thrill and a remarkably loud murmur, *which continued without intermission*, though louder during the arterial pulsations. On dissection, the artery was found to be nearly as large as the abdominal aorta, and its coats not only fragile, but so thin as to resemble a vein rather than an artery.

Remarks.—On the first view of this case, the murmur would be ascribed solely to the state of the artery; but I suspect that the continuous part of it was venous, in consequence of the vein being compressed by the enlarged artery.

Aneurism in the substance of the left auriculo-ventricular septum; disease of the aortic valves, probably with regurgitation: dilatation of the heart.

James Brown, æt. 27, a tailor; complexion cadaverously pale, admitted into St. George's Hospital under Dr. Chambers, Dec. 9, 1829. Palpitation; vehement impulse; throbbing of the carotids; œdema pedum; dysentery; pulse 130, full, strong and *jerking*.

Is a drinker. Has been short-winded for a year at least, and disabled for three months. Died Jan. 15, 1830.

Autopsy.—Left ventricle dilated, but the parietes of natural thickness. *Aortic valves.* Their bases in parts detached by steatomatous disease. Beneath the valve nearest to the left auricle, the little finger could be introduced and insinuated under the lining membrane of the heart to the extent of half an inch, when it emerged through a rugged, steatomatous opening into the cavity of the ventricle. From this canal, a second extended transversely to the left, into the muscular substance of the septum between the auricle and ventricle; and here it formed a pouch about as large as a nut, which bulged upwards and backwards, behind the pulmonary artery.

Remarks.—This case presents an instance of real aneurism, *i. e.* partial dilatation, of the heart. Its physical signs are a desideratum. I did not see the patient before death, and therefore had not an opportunity of noticing them. The detached state of the aortic valves, put in connexion with the jerking pulse, renders it almost certain that there was a murmur from regurgitation. This, however, is not a sign of the aneurism in particular.

The two following highly interesting cases were sent to me by my friend Dr. Lombard, an eminent Genevese physician, educated in England, and in great estimation amongst the English residents at Geneva.

Hypertrophy and dilatation; universal adhesion of the pericardium, with double impulse. Tubercles of lungs, pleura, pericardium, heart, bronchial glands and peritoneum.

A Genevese, æt. 8, had for several months experienced frequent paroxysms of cough and become very emaciated and feeble. When visited, the symptoms were, extreme emaciation; frequent cough; easy expectoration of stringy mucus with yellowish puriform flakes; excessive palpitation; pulse very frequent.

Physical signs.—Impulse. The ear is raised by the beats of the heart, which are tumultuous and of great energy.

Lungs.—Resonance of the left side in front almost flat; rather obscure behind: very clear on the right side. Mucous râle in some parts, especially on the right, where respiration is puerile. On the left, absence of respiration in the greater part of the lungs.

In two months he died from oppression and obstinate vomiting. Two days before death he had suffocative dyspnœa, with the singular phenomenon that the beats of the heart were very intense, tumultuous, and *twice as frequent as the pulse*, the heart pulsating about 150 or 160 per minute, while the pulse beat only eighty.

Autopsy.—The *right lung* contains crude tubercles over a great extent. Pleura sound. *Left lung* almost universally adherent to the ribs by thick tuberculous false membranes covered with albuminous flakes: less numerous but more advanced tubercles than in the right lung: some beginning to suppurate.

Pericardium universally adherent to the heart by false membranes from one to three lines thick, which contain numerous tubercles, in all respects similar to those of the pleuritic false membrane. Heart three or four times as large as natural—hypertrophy of both ventricles, but particularly of the left, of which the walls are very thick and the cavity considerable. In the substance of the walls of the right ventricle is a tuberculous tumor of six or eight lines in thickness, and two or three inches in length, formed by a yellowish resistent tissue more homogeneous at the border than in the centre. Besides this principal tumor there exist several smaller, but in the right ventricle exclusively. Orifices and great vessels healthy. The base of the heart and the origin of the great vessels is encircled with a considerable mass of tuberculous glands, which completely envelope the pulmonary artery and aorta for several inches from their origin. The glands are formed of a yellow, firm, resistent, tuberculous substance: one alone is softened. By their agglomeration they form an irregular mass of several inches in diameter.

Peritoneum granular. Mesenteric glands, also liver, spleen, and intestines, healthy.

Remarks.—This case is remarkable for the prevalence of the tubercular diathesis. I have little doubt that the two beats of the heart for one of the pulse were nothing more than the impulse and back-stroke, which, as in the case of May, become very sensible and have a jogging character when there is universal adhesion of the pericardium.

Immense aneurism of the aorta in the substance of the left lung producing hæmoptysis. Amaurosis.

Lafin, cook, æt. 58, athletic, has enjoyed good health until lately, has been seized within six weeks with head-ache and complete amaurosis of the right eye, and incomplete of the left: pupils are contracted and immovable. Complains of pain equally in the back and the shoulders, by which he is obliged to remain seated in bed. Pulse natural; appetite natural; neither cough nor expectoration. Six weeks later, he begins to cough and to expectorate considerable quantities of blood: the cough returns in paroxysms, and almost always brings a considerable quantity of scarlet and almost pure blood. Respiration is feeble over the whole right side, particularly high up. Heart presents signs of dilatation of the right side. Pulse full, frequent and smart.

He was blooded and cupped several times: the blood being always buffed. The hæmoptysis continued and became black like prune-juice, and was accompanied with extensive mucous râle over the whole left side. Respiration hurried, incomplete. Died on the 17th day of the hæmoptysis.

Autopsy.—Extremely capacious chest, no emaciation, the left lung is universally adherent to the costal pleura by a thick fibro-cellular membrane infiltrated with serum. Its superior lobe contains an aneurismal sac of the size of a cocoa-nut. The sac is formed by the ascending aorta two inches above its escape from the pericardium: *its internal surface is smooth:* the internal membrane of the artery may be traced over a breadth of about two fingers: the rest of the sac is formed by the middle coat, which presents linear furrows that do not alter the polish of the surface. Outside of the sac are found fibrinous layers, less organized in proportion as they are more external: the last, which are in contact with the lung, seem formed by coagulated blood alone.

After having removed the aneurismal sac, the superior lobe of the lung is found reduced to a mere membrane composed of condensed pulmonary tissue: the air vesicles, the bronchial tubes and the blood vessels open directly on the internal surface of the covering of the aneurismal sac, and are thus in immediate contact with the tumor, which, by its size, has caused absorption of a great part of this lobe.

Beyond the aneurism, the aorta is dilated as far as within three fingers breadth of the cœliac trunk: its internal surface is rugous over the whole of this extent, and some cartilaginous points are observed in the thoracic portion.

Heart tolerably voluminous; all its cavities dilated. No contraction of orifices.

The inferior lobe of the left lung is infiltrated with pus, and presents several lumps of ramollissement: some softened tubercles in that part of the superior lobe which is in contact with the tumor; none elsewhere.

The right lung is gorged with serum, and presents several lumps of gray ramollissement. The bronchi are red and tumified.

The second dorsal vertebra presents a commencement of caries, the left part of its body being destroyed over an extent of three lines at the point corresponding with the aneurismal sac.

Opacity of the arachnoid coat and of the pia mater: serum between these two membranes.

Slight atrophy of the left optic nerve after the decussation.

Remarks.—Could so large an aneurismal tumor have been produced in the short space of ten weeks, especially considering that the middle arterial coat extended throughout the whole tumor and the internal over a considerable portion? If it existed previously, it is remarkable that the patient should have enjoyed robust health, without either cough or expectoration.

For ten more cases of aneurism of the aorta by the author, the reader is referred to the London Medical Gazette, September 5th and 12th, 1829.

The following case is a curiosity, as it presents a greater number of different murmurs, (namely, six, including that rare one—the direct mitral,) than I have heard in any other instance: yet it will be seen that they were unravelled with the greatest clearness by a student! This gentleman was Mr. James Freeman, a pupil of my class on the practice of medicine, who brilliantly won my prize for auscultation for the year. I give the case in his own words, the accuracy of which I have verified by a personal examination of the patient.

Aneurism of the aorta: aortic regurgitation: mitral contraction and regurgitation, with two murmurs.

"*John Goff*, aged fifty-five years, in St. Bartholomew's Hospital, May 4, 1839.

"*History.*—About nine months ago, had a violent blow on the right side of the chest from the collar of a horse: did not feel much illness at the time; but, about a fortnight afterwards, was attacked with hœmoptysis, and coughed up blood "by the teacupful."

"*Present symptoms.*—1. The face is very pallid. 2. He has had no hœmoptysis for the last seven weeks. 3. When admitted he was compelled to lie on the right side, but he can now lie on the back, or on either side. 4. The anterior and superior part of the right chest is dull on percussion. The dulness is complete between the third and fifth ribs; it is less complete above the third rib, but it evidently exists as high as the clavicle.

5. The respiratory murmur is deficient over the part that is dull, the deficiency being slight between the third rib and the clavicle. 6. The respiration is slightly puerile in other parts of the chest. 7. There is an obvious prominence of the right side, at the part where the dulness is complete. 8. There is a strong pulsation between the third and fourth ribs about one inch and a half to the right of the sternum. 9. The pulsation is occasionally, but not constantly, accompanied by a purring tremor. 10. There is no preternatural pulsation above the clavicles. 11. There is no tremor above the clavicles. 12. The impulse of the heart is natural.

13. The *first sound* is accompanied,

a. With a near, loud, and slightly rough murmur, loudest between the second and third ribs, about an inch to the right of the sternum; which is heard to a considerable distance, but with diminishing intensity as we depart from that place.

b. Over the apex, a murmur is heard with the first sound, loudest in that situation, and rapidly diminishing as the stethoscope is applied above the apex.

c. Over the humeral ends of the clavicles there is a near and sharp murmur, probably generated in the subclavian artery.

14. The *second sound* is accompanied,

a. With a soft and very prolonged murmur, heard loudest about the lower edge of the third rib, at the right margin of the sternum. It is heard with diminishing intensity below this, along the right margin of the sternum. It is decidedly louder on the right than the left side.

b. The second sound is accompanied with a prolonged, soft, sawing murmur, about the pitch of the whispered letters *awe*, over the apex. It is heard in by far its greatest intensity over the apex, and is greatly and palpably diminished (almost lost) on applying the instrument an inch or two above that place.

15. The sounds are slightly audible on the back. 16. The pulse is eminently "jerking", and slow (60); it is accurately expressed as "celer et infrequens." 17. There is no venous murmur. 18. The patient has never had rheumatism, and cannot give any account indicative of his having had a diseased heart previously to the accident with the horse.

Diagnosis.—1. Aneurism of the aorta. 2. Regurgitation through the aortic valves, or from the aneurism into the right ventricle. 3. Contraction of the mitral orifice, or some obstruction to the ingress of blood through the orifice. 4. Regurgitation through the mitral valve."

Remarks on Mr. Freeman's case. The signs up to 13, inclusive, indicate an aneurism of the ascending aorta. The lowness of its situation denotes that it springs from near the origin of the vessel,—whence, it probably implicates the aortic valves, as in fig. 13. The murmurs over the humeral ends of the clavicles are subclavian; but, over the sternal ends, they had an abrupt, hoarse intensity, which is usually connected with disease of the interior of the aortic arch.

The murmur *a*, with the second sound, coinciding with the eminently jerking pulse, indicates regurgitation through the aortic valves. This murmur is heard more to the right than usual, in consequence of the aorta being a little displaced in that direction by the aneurismal tumor, which, when impacted between the sternum and spine, generally slips to the right, where there is less pressure. Mr. Freeman puts the alternative of the regurgitation being "from the aneurism into the right ventricle." This is a fair and shrewd alternative, and it is drawn from a supposed analogy with the case of Mitchell, p. 466, and fig. 21. This view, however, is discountenanced by the facts, 1. that, in the case of Goff, the murmur was that of an ordinary semilunar regurgitation, whereas, in Mitchell, it was anomalously loud, rough, and continuous; 2. that, in Goff, there was no venous lividity from intermixture of arterial and venous blood; and 3. that there was no dropsical tendency, which symptoms were highly marked in Mitchell (see Signs, p. 471).

The two murmurs at the apex denote the mitral contraction and regurgitation; but the regularity of the pulse and the continuance of its jerk, indicate that they are not very great. The prolonged character of the direct mitral murmur—in short, its close analogy to the murmur of semilunar regurgitation, is well worthy of remark. I have not noticed this character in any other case. It remains to be ascertained by further observations, whether it is constant.

DESCRIPTION OF THE PLATES.

FIG. 1 (the frontispiece) illustrates the description given at p. 2, of the situation of the heart and great vessels with respect to the exterior. The patient is supposed to be horizontal: when erect, the heart is a little lower. The Fig. also illustrates the situation of the jugular veins, vena innominata, and carotid arteries, described at p. 112, in reference to venous murmurs.

a. The internal jugular vein, running in front of the carotid artery, along the anterior margin of the sterno-mastoid muscle, immediately beneath the teguments and platysma myoides.

b. The external jugular vein.

c. Oblique section of the sterno-mastoid muscle, which crosses the internal jugular vein at its lowest part.

d. The thyroid gland.

e. The trachæa.

f. The arteria innominata.

g. The left carotid.

h. The subclavian artery.

i. The clavicle, cut short.

k. The vena innominata.

l. The arch of the aorta.

m. The pulmonary artery.

n. The right auricle.

o. The right ventricle.

p. The appendix of the left auricle.

q. The left ventricle.

The sternum and ribs are represented in dotted outline. The ribs are numbered 1, 2, &c.

FIG. 2.—This and the following figure are introduced for the purpose of showing the exact situation and mode of action of the auricular valves and their columnæ carneæ—a subject little understood, and of which I have seen no good plates. I selected the most healthy heart with which I could meet, (from an adult female of average size,) and made fac-simile drawings.

a. The aorta.

b. Pulmonary artery.

c, d, e. Three pulmonic valves, *d* being central and posterior.

f. A muscular prominence, from which proceeds a great number of very fine tendinous chords to the anterior lamina of the tricuspid valve.

g. The anterior lamina of the valve, strengthened by tendinous chord radiating in a fan-like manner from the columna carnea, *i.*

h. The third principal division of the valve, partly anterior and partly posterior, springing from a columna carnea, behind the one *i*, and sending a long tendinous chord upwards to the posterior lamina of the valve.

i. A columna carnea which, with a portion of the ventricular wall *l*, has been separated from the cut edge *m*. The columna springs posteriorly from the septum ventriculorum, and naturally draws nearly in the direction *g m*.

k. Six fine tendinous chords springing from the posterior part of the septum, and going to the posterior lamina of the valve.

l. Cut edge of the ventricle corresponding with the edge *m*.

Remarks.—The valve and its columnæ carneæ are situated entirely at the posterior side of the ventricle, so that, when the blood has entered, it is wholly in front of them. During the ventricular contraction, the blood presses the two laminæ of the valve not only flat against each other, but also against the posterior wall of the ventricle; whence the valve is entirely withdrawn from the current of the blood, and presents no obstacle to its egress through the pulmonary artery. It is, in short, strictly an oblique valve, shut by its own contents, like the valves of the ureters.

The objects of the columnæ carneæ are, 1. gently to draw down the upper lamina of the valve after the diastole, preparatory to its complete occlusion by the pressure of the blood in front during the systole: 2. to hold the valve firm, and prevent its being forced back into the auricle, during the systole. This is probably assisted by a contractile shortening of the columnæ carneæ, which maintains the valve in situ by countervailing the contraction of the apex towards the base—a movement tending to give too much latitude to the valve.

As the columnæ carneæ spring from near the apex, it is obvious why the sound of the valve is better transmitted to this part than to the front of the base, opposite to the orifice, where the sound has to be transmitted through the mass of blood in front of the valve. This illustrates what is stated at p. 64 and 91 respecting the situations in which to explore the sounds of the auricular valves.

Mr. King and others have imagined that the tricuspid valve naturally admits of regurgitation, and, in so doing, exercises a safety-valve function. It is credible that it may *shut out* any redundancy of blood, but I do not believe that, when once closed, it admits of regurgitation: 1. because the structure of the valve is, in my opinion, distinctly opposed to such a doctrine; 2. because no murmur attends the first sound—an argument which alone would be conclusive to an auscultator.

FIG. 3 represents the interior of the left ventricle and the mitral valve.

a. Aorta.

b. Interior of the pulmonary artery.

c. Portion of a pulmonic valve, cut from——

d. The remaining portion.

e and *f.* The two other pulmonic valves. It is thus seen that the pulmonic valves are seated about half an inch higher up than——

g. The aortic valves.

h. Anterior lamina of the mitral valve, fully half an inch broad, and situated almost immediately below the central and posterior aortic valve *g.*

i. A columna carnea sending its chordæ tendineæ over a probe to the anterior lamina of the valve, on which they radiate.

k. Another columna carnea sending its chordæ in a similar manner to the other corner of the anterior lamina. When the ventricle is in its natural closed state, this columna falls into the shady depression *n* between *k* and *i*, and is almost in contact with the columna *i*.

l. Posterior division of the columna *i*, sending its chords to the posterior lamina of the valve, which is only about a quarter of an inch broad.

m. A second posterior columna sending its chords to the posterior lamina. This columna is, in the present instance and in Fig. 20, a part of the mass *i*; but, in other instances, as Figs. 5 and 7, it forms the posterior division of the mass *k*.

n. The depression into which the columna *k* falls.

o. Section of the ventricular walls, made close to the septum, and passing through the apex.

Remarks.—It is seen that the valve and columnæ are situated entirely at the posterior part of the ventricle, and that the columnæ draw almost straight downwards towards the apex. The mode of action of the valve, and the transmission of its sounds to the apex, are exactly the same as in the case of the tricuspid valve (see Fig. 2, Remarks).

Mr. Mayo appears to have been the first who pointed out the principle upon which the auricular valves close their respective orifices (see Med. Gaz. Aug. 10, 1833, p. 635).

FIG. 4. A. B. and C. It is suggested to the young student to carry these three diagrams, with their descriptions, in his pocket, till he is master of the subject. They illustrate the descriptions given in the sections p. 70 and 90, where further details will be found. They also apply to the summary of the physical signs of valvular disease at p. 383, et seq.

Each of the four orifices of the heart may, by disease of its valves, be the seat of two murmurs,—one, from the blood flowing in its natural direction; the other, from its regurgitating or flowing retrograde through the permanently open valve. The former murmurs may be called *direct;* the latter, *regurgitant.*

They are represented by the two following diagrams, which apply equally to both ventricles, though drawn in reference to the left only.

Diagram 4. A. The ventricle is supposed to be in the state of systole.

a. Is a *direct* aortic or pulmonic murmur.

b. Is a *regurgitant* mitral or (in the case of the right ventricle) tricuspid murmur.

Diagram 4. B. The ventricle is now supposed to be in the state of diastole.

a. Is a *regurgitant* aortic or pulmonic murmur.

b. Is a *direct* mitral or tricuspid murmur, (which is extremely rare, often absent though the valve be contracted, always feeble, and, in the case of Goff, p. 611, it was prolonged like a semilunar regurgitant murmur, but I am not yet sure that this character is constant).

Diagram 4. C. represents the situations where the murmurs of the respective valves are most audible, and affords the principal data for the differential diagnosis.

a. About two inches up the aorta.

b. About two inches up the pulmonary artery.

c. Over the two sets of semilunar valves, at the level of——

d, *d*. The inferior margin of the third rib, when the patient is horizontal. When he is erect, the valves are drawn a little lower down.

e. The right auricular orifice (see Fig. 2).

f. The right columnæ carneæ and chordæ tendineæ.

g. The left auricular orifice (see Fig. 3).

h. The left columnæ carneæ and chordæ tendineæ.

Differential diagnosis of diseases of the semilunar valves.—A murmur with the first sound at *c*, if distinct at *a*, is aortic; as a pulmonic murmur is only feebly transmitted, and on a lower key, in that direction.

A murmur with the first sound at *c*, if distinct at *b*, is pulmonic; as an aortic murmur is only feebly transmitted, and on a lower key, in that direction.

A prolonged murmur with the second sound, loudest at *c*, is from semilunar regurgitation. It is aortic, if the murmur is loudest in the direction *a h*, gradually diminishing on descending from *c* down the ventricle. It is pulmonic, if loudest in the direction *b f*, gradually diminishing on descending from *c* down the ventricle.

A murmur with either sound, if distinct at *a* or *b*, is semilunar; as an auricular murmur is either inaudible or very feeble, and on a low key, so far off.

Differential diagnosis of diseases of the auricular valves.—A murmur with the first sound, loudest at *h*, (which is about where the apex impinges, and a little to the sternal side of the nipple,) is from mitral regurgitation: as a tricuspid regurgitant murmur is comparatively feeble in that situation.

A murmur with the first sound, loudest at *f*, (which is about the same level as *h*, but under the sternum,) is from tricuspid regurgitation; as a mitral regurgitant murmur is comparatively feeble in that situation.

A murmur with the second sound, loudest at *h*, is from contraction of the mitral valve: one, loudest at *f*, is from contraction of the tricuspid.

A murmur with either sound, loudest at *h* or *f*, is auricular; as a semilunar murmur is very weak, on a low key, and sometimes wholly inaudible, so far off.

FIG. 5. Thickening and contraction of the mitral valve, with thickening and shortening of the chordæ tendineæ.

a. b. The anterior lamina, cut through its middle into the auricle, and held open by two threads, to display the posterior lamina *c. d.* which presents a thickened ridge. The two laminæ are agglutinated together at their extremities *d* and *c*; whence the total circumference is diminished, being only 2⅞ inches instead of 3½ (see p. 295).

e e and *f f* are the two double masses of columnæ carneæ, the anterior sending chord to the anterior lamina, and the posterior to the posterior. The chords, especially the anterior, are seen to be considerably thickened and shortened.

Remarks.—When the chordæ tendineæ are shortened, as in this instance, they cause the superior lamina of the valve to be held slightly open by the columnæ carneæ, when all the parts are put on the stretch during the ventricular systole. The mechanism of this will be very evident to one who has studied Figs. 2 and 3. Yet it is a lesion almost constantly overlooked. The regurgitation is favoured by prominences on the edges or expansions of the laminæ, (as the ridge *c d*,) preventing their close apposition; for the blood then insinuates itself between the laminæ by a wedge-like process.

The patient, *Charles Porter*, æt. 18, was in St. George's, March 15, 1836. A murmur with the first sound was heard very distinctly towards the apex (mitral regurgitation). Communicated by Dr. Nairne.

FIG. 6, represents a great degree of fibrous thickening and contraction of the aortic valves, from endocarditis. A lump of this fibrous tissue, as large as a pea, existed in the inside of the central valve, and raised it externally at *a*. I did not see the patient during life.

FIG. 7. This is introduced to exhibit a greater degree of thickening and shortening of the chordæ tendineæ and contraction of the aperture of the valve, than Fig. 5.

a. a. The right columna carnea, consisting of the anterior and posterior divisions,—each sending chords to the corresponding lamina of the valve.

b. A greatly thickened and shortened mass of agglutinated tendinous chords, going to the anterior lamina. *c.* Posterior chords.

d. d. The left columna carnea, which has been cut from its origin *e, f,* and drawn aside. It consists of the anterior and posterior divisions,—each sending short and thick chords to the corresponding lamina.

e. f. The point from which the columna *d d* has been cut.

g h. Chords going from *d d* to the anterior and posterior laminæ.

i. Boundary of the arterior lamina.

k k. Aortic valves.

Remarks.—The valvular aperture was contracted by thickening, and by the agglutination of the ends of the two laminæ, so as barely to admit the point of the first finger.

"A blowing first sound was heard over the region of the mitral valve—most distinct towards the apex" (Dr. Nairne). The patient was Peter Crump, in St. George's, March 22, 1836.

FIGS. 8 *and* 9. *a.* The arteria innominata, cut open.

b. Orifice of the subclavian.

c. A thickened and steatomatous flap of the internal membrane, moveable up and down, so that, when up, it shuts back upon the orifice of the subclavian *b,* and closes it like a valve—whence this artery and its branches were pulseless.

The lesion originated in steatomatous disease, which had led to a rupture and dissecting up of the internal membrane. The interior of the whole ascending aorta and arch was exceedingly steatomatous, and the vessel was dilated to about three times its natural size. At one point it had formed a pouch, which bulged against, and finally burst into the lungs, and was fatal by hæmorrhage. The patient was in St. George's, Sept. 19, 1837.

FIGS. 10 *and* 11. The angle (*a.* Fig. 11) of one of the aortic valves torn from its origin, and the flap hanging back, so as to admit of regurgitation, which was attended with a loud musical murmur. Fig. 10, *a,* represents a perforation in the same flap.

The other valves are thickened. See case of Milton, p. 587, and musical murmurs, p. 87.

FIG. 12. Contraction of the mitral valve, from hypertrophy of the fibrous tissue and adhesion of the two laminæ, with shortening and thickening of the tendinous chords, whence regurgitation and murmur.

a and *b*. The two divisions of the right columna carnea, the division *a* being cut short and turned up.

c. The two divisions of the left columna, cut short and held out.

d. Left auricle.

FIG. 13. *a*, an aneurism of the aorta as large as a bantam's egg.

b, *c*. The edges of two valves, depressed by the aneurism, thickened by fibro-cartilage, and everted, whence free regurgitation and a murmur.

d. Third valve, much contracted.

e. *e*. Steatomata. (See case of Williams, p. 583.)

FIG. 14. Fibrous thickening of the aortic valves, especially the valve *c*. It occasioned a direct, and a regurgitant murmur. In St. George's, May, 1838.

FIG. 14. *A*. Extraordinary disease of the aortic valves. They hung loosely back into the ventricle, not offering the slightest resistance to the blood. The valves *a* and *c* were thrown together into one large, irregular flap. *b* is a rugged lump of mixed bone and steatoma, very crisp and rotten. Similar bone edges the dependent flap *e*. The membranous expansions of the valves were greatly thickened by fibrous tissue. *f* and *g* are the two coronary arteries. Patches of yellow steatoma, *h h*, are seen at the origin of the aorta. The mitral valve *d* is healthy.

James Windsor, æt. 33, out-patient at St. George's, June 20, 1838. A double aortic murmur. P. 80, jerking. *Diagnosis*. Aortic contraction and regurgitation. No mitral disease.

July 2. Pain in right side last week. Side dull over lower half; no respiratory murmur, crepitant râle, or fremitus; ægophony: dry cough. *Diagnosis*. Acute pleurisy with effusion. He did not return, but died.

The gentleman who made the examination informed me that I had committed an error in diagnosis, the aortic valves being sound and the mitral diseased. I ventured to express myself incapable of being convinced, except by seeing the preparation. It was sent. He had mistaken the aortic for the mitral valve. I have so frequently seen similar mistakes, that I never hold myself responsible for any diagnosis not given in writing, and of which I do not personally witness the post-mortem results.

FIG. 15. *a.* The three aortic valves, stiffish with calcareous scales of opaque yellowish-white colour. A ring of bone as thick as a quill encircles the aortic orifice at *b*, but is concealed by the lining membrane. A similar ring, *c*, equally thick, encircles the base of the mitral valve. In parts, it is denuded and rough: elsewhere, the lining membrane invests it like a blue film.

The patient was in St. George's, under Mr. Babington, and had attained the age of 80 without complaining of symptoms of disease of the heart. The aorta and coronary artery were also ossified. This case is referred to at p. 261.

FIG. 16. *a* and *b* are rough calcareous concretions from the aortic valves: *c* was a smoother one. They projected into the vessel, and caused loud rasping murmur. The reasons are explained at p. 82. The pulse was little affected in fulness, firmness, and regularity. See p. 378.

FIG. 17. The aortic valves seen from the ventricular side. They are agglutinated together, and form a fibro-cartilaginous ring, *a*. See case of Hedgley. The pulse was small, weak, and unequal, for the reasons of which see p. 378.

FIG. 18. A dried preparation of ossified aortic valves, viewed from the aortic side. The valves *c*, are curled and contracted, and project in the closed position into the interior of the vessel, so as greatly to contract the orifice. *a* are two flaps of the arterial walls peeled off *b*, to show that the valve and its base are converted into one thick, osseous mass. The surface of the concretions is everywhere granular and rough.

FIG. 19. Steatomatous and calcareous disease of the interior of the aorta. *a*, the opening into the arteria innominata: *b*, into the left carotid, thrown much to one side: *c*, into the left subclavian. All are much contracted. This Fig. illustrates p. 419.

FIG. 20. 'An ossified aneurism as large as an egg, in the muscular substance of the left ventricle, communicating with the aorta by an aperture as large as a swan-quill, under the base of one of the sigmoid valves. The aperture originated in steatomatous disease. A stick *a* is passed through it from the aorta *b*, and emerges by a second opening, *d*, *e*, into the cavity of the ventricle. The latter opening is enlarged by a slit to the right, better to display the interior of the aneurism, the walls of which are perfectly hard and rigid, except at the slit, *d*, where they are fibro-cartilaginous. The bases of the other sigmoid valves, *c*, *c*, are thickened and elevated by steatomata. *f* is the anterior, and *g* the posterior, lamina of the mitral valve; *h* is the right columna carnea, which is triple; *i* is the left mass, which is single; both are exceedingly hypertrophous. *k* is the wall of the ventricle an inch and a quarter thick.

Remarks.—This case is referred to at p. 380. It is also an excellent instance of hypertrophy. The patient was an old woman in the St. Marylebone Infirmary, who laboured under aggravated symptoms of organic disease of the heart. I did not see her during life. The physical signs were not noticed.

FIG. 21. Aneurism of the aorta bursting into the right ventricle. *a*, the pulmonary artery: *b*, an interval between the two pulmonic valves *c* and *d*, through which regurgitation could take place; *e*, two apertures through which the aneurism discharged the aortic blood into the right ventricle; *f*, cavity of the ventricle.

See the remarkable case of John Mitchell, p. 466; and the signs, p. 471.

FIG. 22. Plan of the degree of excavation suitable for the ear-piece of a stethoscope. It suits almost every ear. See p. 93, note. This is beyond comparison the most important part of the instrument. The nature of the wood is of trifling importance, though, theoretically, cedar is the best.

TABLE OF PULSES OF DISEASE OF THE HEART.

(*See Remarks, pages* 375–6.)

Simple Hypertrophy of Left V.—*Strong* and *tensely prolonged;* because the ventricle contracts powerfully but slowly. (p. 265.)

Hypertrophy with Dilatation.—*Strong, tensely prolonged,* and *large;* because the ventricle contracts powerfully, slowly, and expels an increased quantity of blood (p. 265).

N. B.—If the above pulses be moderately accelerated, they become "*Hard.*" They may be rendered temporarily or permanently *small and weak* by any debilitating causes, impairing the contractile power of the heart. Also, by extreme palpitation and dyspnœa causing engorgement of the organ.

Hypertrophy with Contraction.—*Tense* but *small;* and if the contraction be considerable, it becomes weak as well as small, from the insufficient quantity of blood propelled into the arteries (p. 265).

Dilatation with Hypertrophy, *i. e.* the dilatation being predominant—*Large* and rather *prolonged,* but *soft;* from the large capacity, but weakness of the ventricle (p. 265).

N. B.—This pulse, if accelerated, becomes "*Bounding.*"

Dilatation with Attenuation.—*Large* and *weak,* becoming *small* in the last stage, when the ventricle is too weak to expel its contents (p. 305).

Softening.—*Small, weak,* and more or less *irregular, unequal* and *intermittent,* sometimes extremely so, in the late stages; from the debility of the ventricle (p. 340).

FREE REGURGITATION THROUGH THE AORTIC VALVES.—Eminently *jerking;* from the arteries being unfilled (p. 379).

CONTRACTION OF THE AORTIC VALVES.—*Strength* little impaired, unless the contraction be very considerable. The *regularity* is seldom affected, except by extreme contraction (p. 378).

GREAT CONTRACTION OF, OR FREE REGURGITATION THROUGH, THE MITRAL VALVE.—*Small, weak, irregular, intermittent* and *unequal;* because contraction occasions an insufficient and irregular supply of blood to the ventricle; and because regurgitation weakens the pulse, in consequence of the resistance of the mitral valve being removed, and disturbs its regularity, in consequence of rendering the supply of blood less uniform (p. 376).

A LARGE POLYPUS FORMED BEFORE DEATH.—Suddenly causes a small, weak, irregular, and intermittent pulse; because the polypus chokes up the ventricle (p. 531).

ENDOCARDITIS WITH POLYPUS.—Ditto, (p. 214).

PERICARDITIS WITH MUCH SEROUS EFFUSION COMPRESSING THE HEART.—Ditto (p. 158).

Remarks.—I hope hereafter to show that these pulses imitate all those produced by ordinary diseases, &c.:—consequently, that, unless the pulses of disease of the heart be abstracted, the pulse is but a fallacious guide in other diseases. As this abstraction, in a complete manner, has hitherto been impossible, in consequence of the imperfect state of our knowledge respecting the pulses of disease of the heart, the present Table is an attempt to supply that deficiency.

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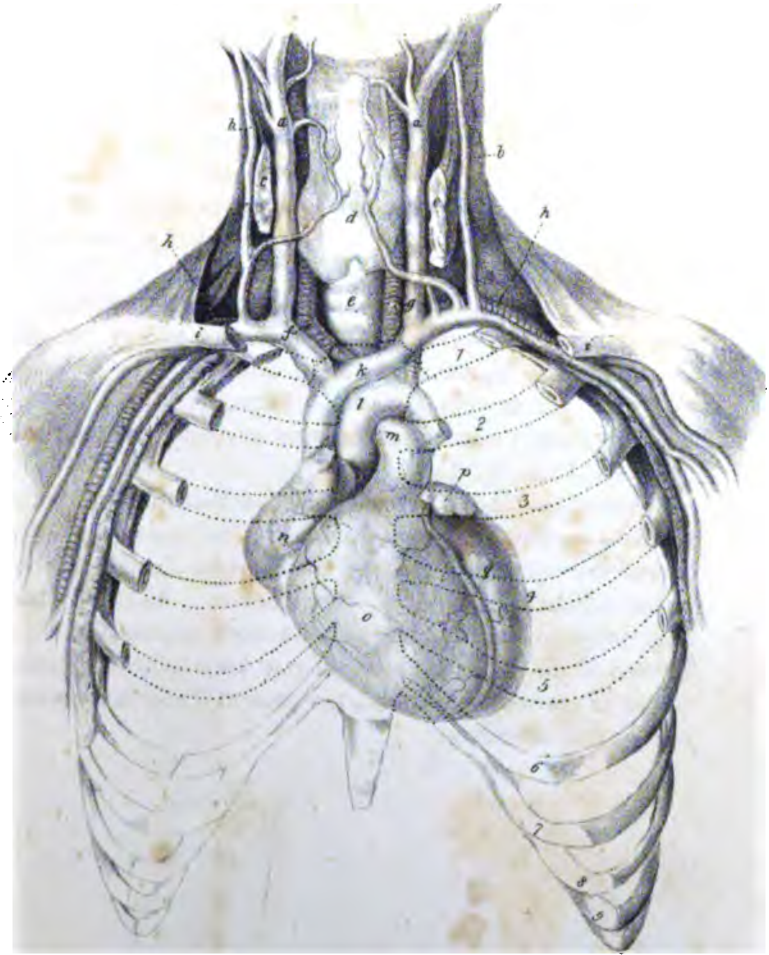

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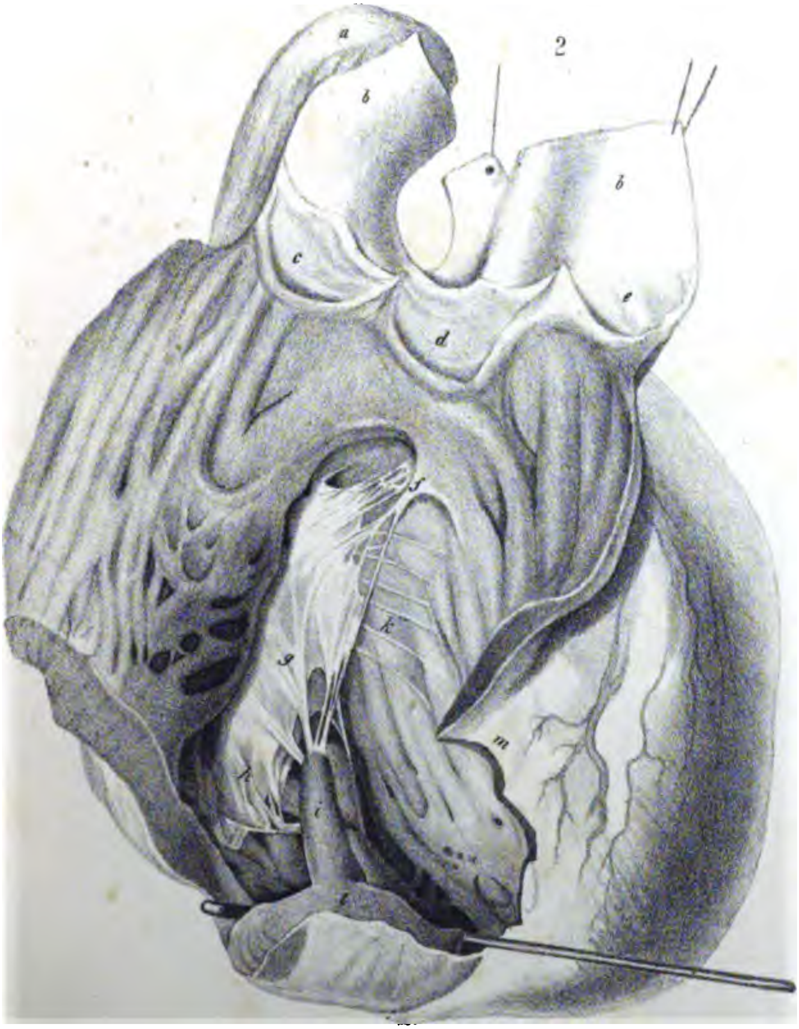

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by the Author.

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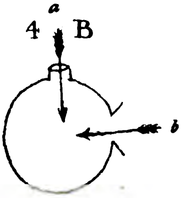

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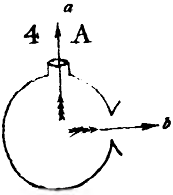

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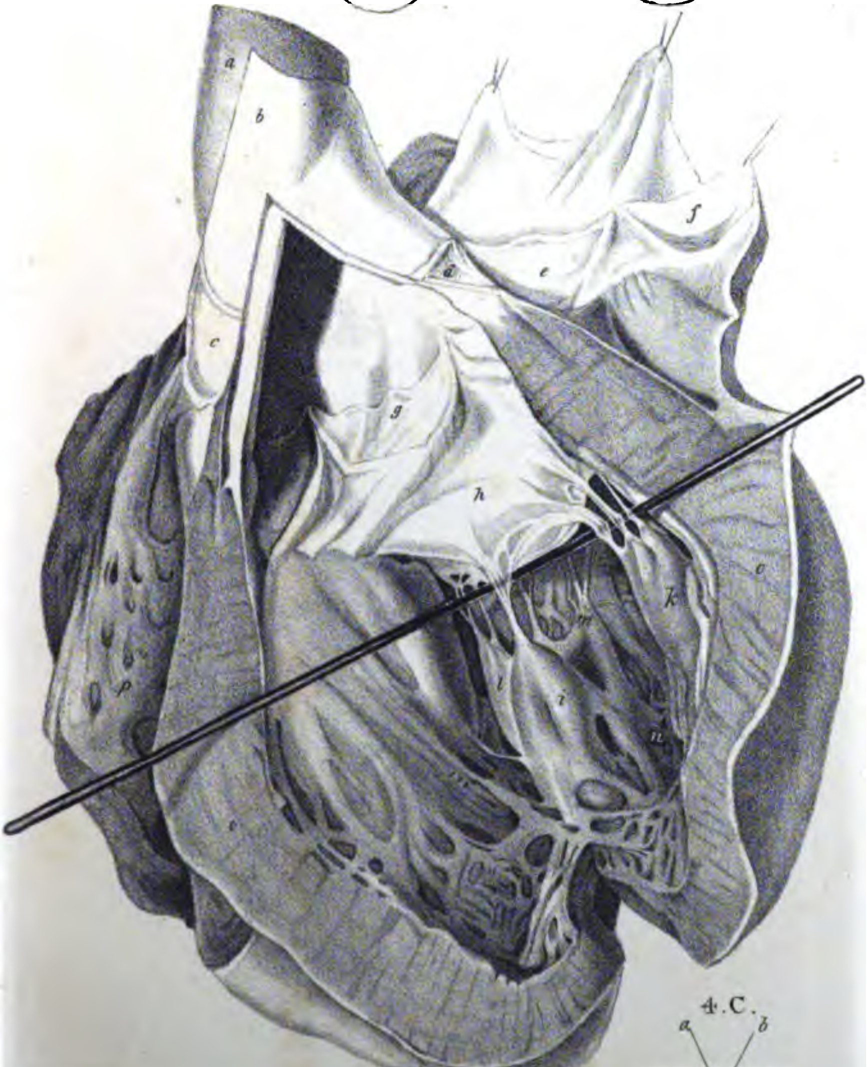

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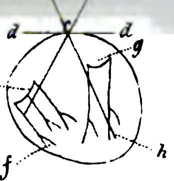

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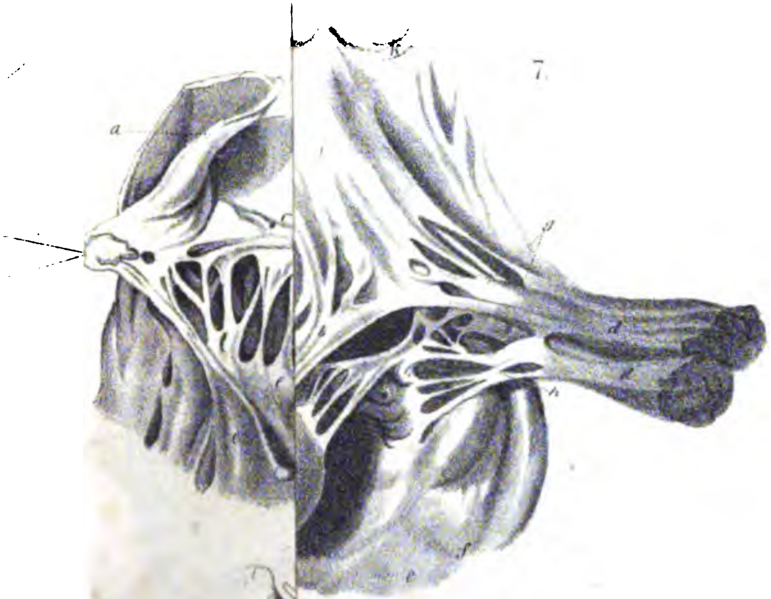

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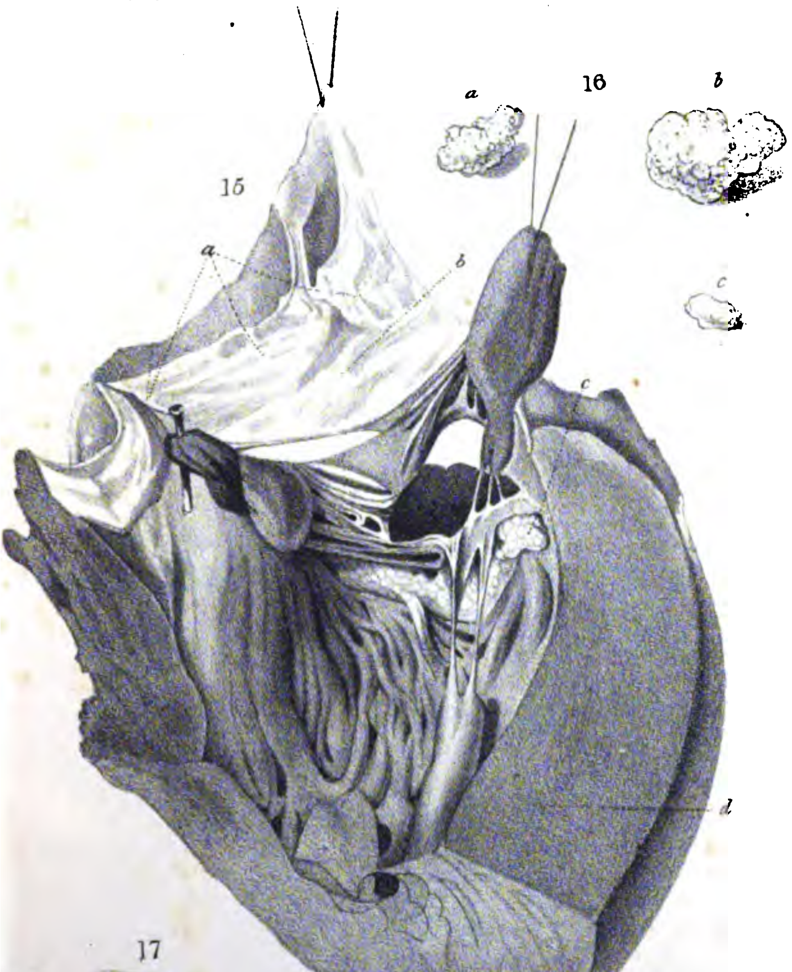
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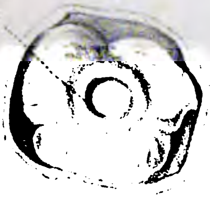
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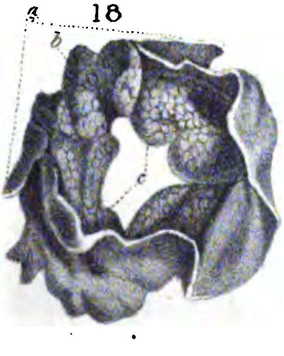
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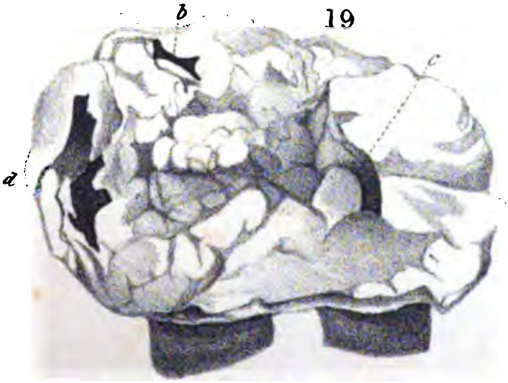
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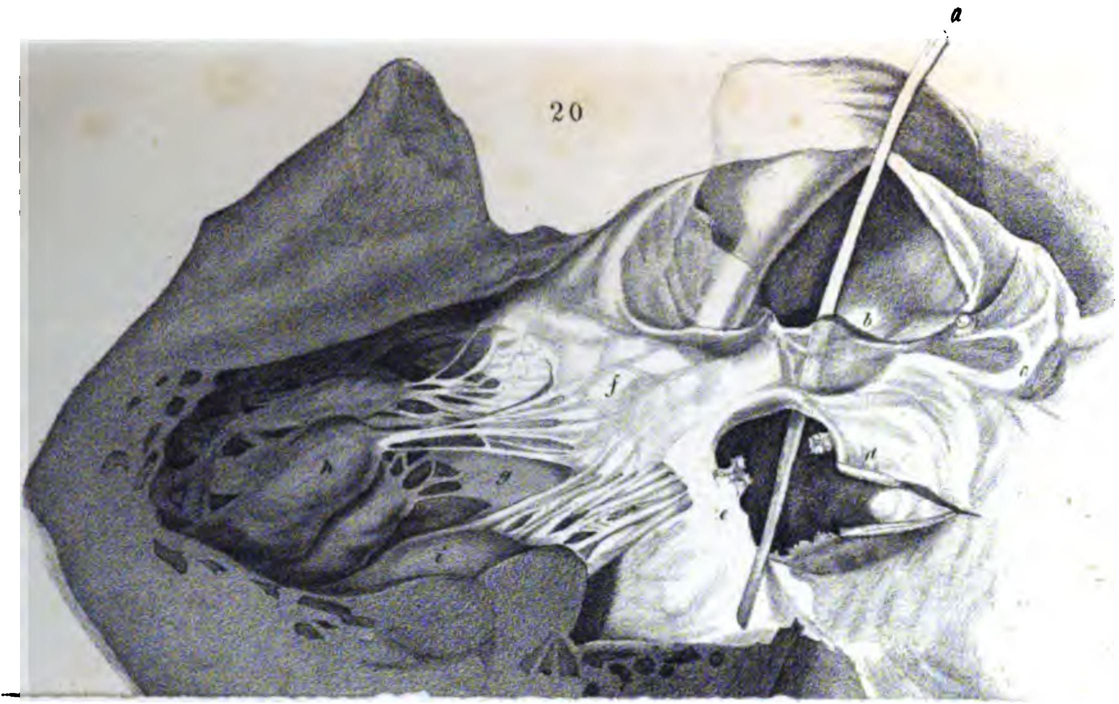

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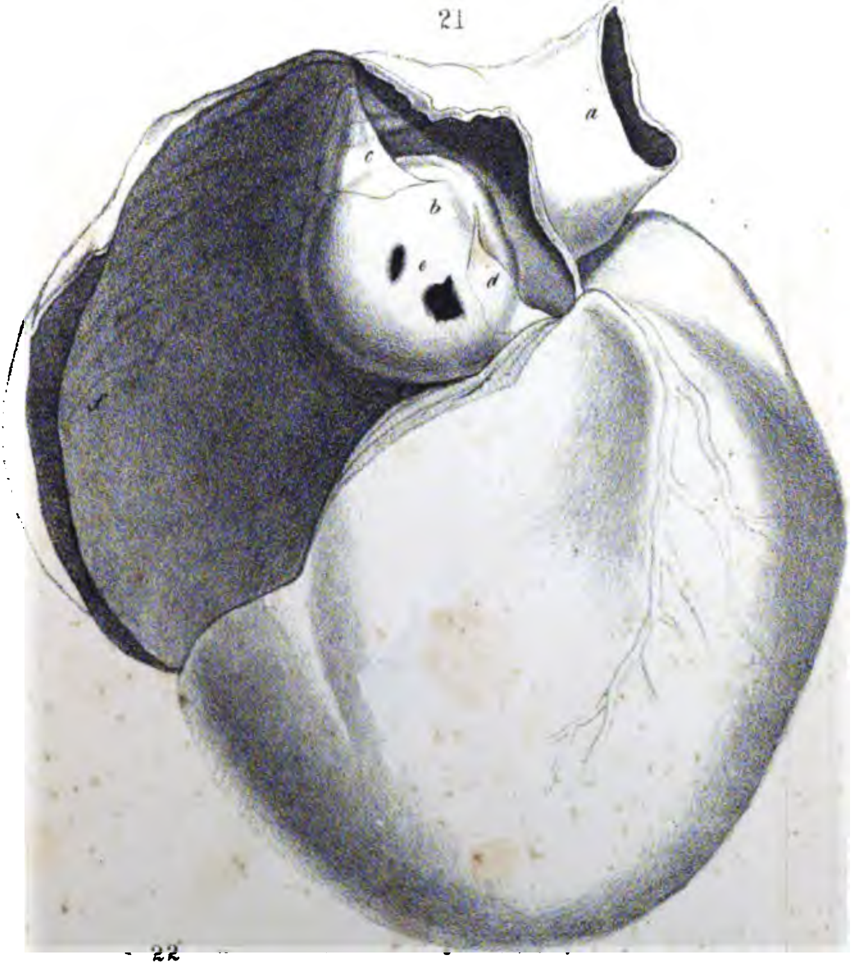

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by the Author.

APPENDIX.

The patients to whom the following autopsies refer, having died after their cases had passed through the press, I insert the morbid appearances, not only as accurately verifying the diagnosis, but as being highly interesting and instructive.

V . . . . , Esq., whose case is detailed at p. 589.

The *Diagnosis* was—"Mitral regurgitation: contraction of the aortic valves and regurgitation: little or no hypertrophy or dilatation."

Autopsy.[*]—The walls of the *left ventricle* were only a little thicker than natural—barely exceeding half an inch, and the cavity was moderately dilated, being about the size of a goose's egg. The walls of the *right ventricle* were of natural thickness, and the cavity was slightly dilated. The *aortic valves* presented the greatest degree of ossification that I have ever witnessed, and I am not aware that a greater has been described by authors. The valves in the closed position, their bases, and the whole circumference of the aorta, were converted, with an exception presently to be noticed, into one solid, immovable mass of bone, retaining the form of the valves and surrounding zone of the aorta, but two to three lines thick, and presenting a scabrous and uneven surface. In the centre, where the three valves meet, was a roundish aperture, two lines in diameter; and from this to the circumference of the aorta was a slit, formed by the unadherent margins of two valves, which were still flexible over an extent of about a line on each side of the slit, and in contact so as to prevent regurgitation, except through the central aperture. The *mitral valve* admitted the passage of three fingers, but its margins were thickened and nodulated. Some of the chords were slightly thickened, and the columnæ carneæ were remarkably thin and pointed, as if stretched from being too short.

Remarks.[†]—The diagnosis was accurately verified. The hypertrophy and dilatation, as anticipated, were not considerable, and they might have supervened during the year which intervened between my examination and his death. The contraction of the aortic valves produced the musical murmur with the first sound over and beyond them, together with the common murmur, which was on a lower key than a whispered *r*, because, as predicted, the circulation through the aorta was weak—a necessary consequence of the extraordinary degree of valvular contraction. The central valvular aperture was the cause of the regurgitation, and, as predicted from the weakness of the murmur, it was not considerable, in consequence of the smallness of the aperture.

The state of the mitral valve was calculated to admit of regurgitation, yet not to a considerable amount.

This case is not only interesting in reference to the two musical murmurs, but important as substantiating the doctrines of the pulse broached at p. 378, with respect to the aortic valves. The pulse was "small, weak, irregular, and unequal." I contend that this (when not dependent on softening) is, as the general rule, the pulse of great contraction of the mitral valve, or free regurgitation through it, and that it is foreign to contraction of the aortic valves, unless extreme. Now, in this case, there was no mitral contraction, and certainly little regurgitation; but there was an almost unexampled degree of contraction of the aortic valves: consequently, the state of the pulse was, it may be fairly contended, a result of the latter.

John Goff, whose case is described at p. 610, died May 27th, and was examined on the 28th. Mr. Freeman had seen him a few days before death, and found the symptoms the same.

The sternum required to be dissected from an adherent fibro-cartilaginous tumour underneath. The third right rib, one and a half inch from the sternum, was slightly eroded over an extent of about half an inch. The sternum itself was also eroded over an extent of one and a half inch long and half inch broad below and opposite to the same rib, the insertion of which was implicated in the erosion. A circular surface of the sternum and ribs of at least two inches in diameter, including the erosions, formed the anterior boundary of an aneurismal sac.

The right pulmonary pleura was firmly adherent to the costal by fibro-cartilaginous tissue, over the upper half of the lung. The cavity below contained seventy ounces of blood (separated into crassamentum and serum). The upper lobe of the right lung was less crepitant and more dense than natural, having the feel of flabby flesh (the carnification of Laennec, from compression).

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[*] The heart was obligingly brought to me, May 24th, by Mr. Eis dell, and is deposited in the museum of St. George's Hospital.

The cavity of the aneurismal sac equalled the size of an average orange. An aperture, through which the handle of a scalpel easily passed, existed at the inferior and posterior part of the sac, and through this the blood had escaped into the cavity of the chest. The sac communicated with the aorta by an irregularly rounded opening about the size of an egg, half an inch above the valves. The interior of the sac was invested by the lining membrane, affected with steatomatous and osseous disease, over about one-half of the posterior and inferior part: over the remainder it was deficient, and thin fibrinous layers supplied its place.

The *pericardium* was universally adherent by old, but lax cellular tissue, which easily admitted of separation by the finger.

The *right ventricle* and its valves were healthy.

The *three aortic valves* were each smaller than natural by fully one-third, so as necessarily to leave an interval between them when in the closed position. The smallness was referable to fibrous thickening, (hypertrophy,) and the membranous parts of the valves were corrugated transversely on themselves, so as to be defective in depth.

The *aorta* above the valves was slightly dilated, and exceedingly rough from steatomatous depositions with a little bone. A similar state existed, but in a less degree, and without dilatation, along the whole arch, and a little beyond. The same also extended up and beyond the innominata, and in a less degree up the left carotid and subclavian. The anterior lamina of the *mitral valve* was fully a line thick, and irregularly nodulated round the edge, from fibrous hypertrophy. The posterior lamina was similarly affected, but in a less degree. The valve admitted of the passage of two fingers easily, though closely, but not of three.

Left ventricle healthy: auricles healthy.

Present, and signed by, HUGH P. FULLER, M.R.C.S.
STEPHEN YELDHAM, M.R.C.S.
THOMAS ABRAHAM, M.R.C.S.L.
THOMAS BLYTH, M.R.C.S.L.
JOHN H. HOUGHTON, M.R.C.S.
FREDERICK DANFORD, M.R.C.S.
JAS. FREEMAN.
G. H. YOUNG.

Remarks.—The diagnosis of Mr. Freeman, and the *remarks* upon it by the writer, were verified in every particular. The condensation of the superior lobe of the right lung was indicated by the defective resonance and respiratory murmur. The aneurism, its origin immediately above the valves, and the diseased interior, without dilatation, of the arch of the aorta, were all correctly indicated. The regurgitation through the aortic valves existed, as anticipated in my remarks. The mitral disease also existed, and, as foretold, was "not very great." The murmur with the second sound in this valve must, I presume, have been occasioned by the contracted state of the valve, (which admitted two fingers only instead of three,) assisted by the nodulated state of the margins, two or three of the nodules being two or three lines thick. Another circumstance, inadvertently omitted in the autopsy, may be noticed. The chordæ tendineæ, at their insertion into the laminæ of the valve, were subdivided and *reticulated* in an unusual degree. It may be asked whether the filtration of blood through the reticulations contributed, with the contraction and nodulation of the valve, to produce the murmur with the second sound. The circumstance is apparently trifling; yet everything should be noticed so long as the precise causes of the murmur in question, and of its frequent absence in greater degrees of disease, are doubtful and under investigation. The regurgitant mitral murmur was abundantly accounted for.

The jerk of the pulse resulted from the aortic regurgitation, assisted by the anæmia. The diagnosis did not include adhesion of the pericardium, and this circumstance corroborated the opinion offered at p. 193-4, that adhesion cannot in every case be predicted with absolute certainty. The reason why the characteristic signs (p. 194) were absent in the present instance, was, that the adhesions were so loose and tender as still to allow considerable latitude of motion; that the heart was pushed back by the aneurism in front, whence any increased or double-jogging impulse was rendered less perceptible: and lastly, that there was no hypertrophy.

I cannot but call attention to the circumstance that this singularly complex case was completely unravelled by a young student, who, three months previously, had no knowledge of valvular diagnosis.

ALPHABETICAL INDEX.*

Abscess in the heart, 197
Acids, use of, in valvular disease, 415
Aconite, use of, in rheumatism, 180
Active aneurism (see *Hypertrophy*)
Adhesion in acute pericarditis, object of, 148
———, process of, 149
——— in chronic pericarditis, 153
——— causes enlargement of the heart, 192, 279
———, danger of, 191
——— may remain after symptoms cease, 164, 181
———, signs of, 169, 193
(See *cases of May, Harrison* and *Williams*.)
Age, a cause of ossification, 227, 258, 363
——, effect of, in hypertrophy, 289
——, effect of, in aneurism, 326
Air, change of, in dilatation, 318
Albertini and Valsalva, their treatment of hypertrophy and aneurism, 282, 356, 474
Anæmia, cause of murmur in hypertrophy, 94
——— inorganic murmurs of heart and arteries, 101
——— venous murmur, 122
——— purring tremor, 126
——— palpitation, 507
——— softening, 337
——— quick pulse after fever, 335
Anæmia, caused by treatment of aneurism, 477
———, caused by treatment of hypertrophy, 283
———, its influence or the pulse of hypertrophy, 265
———, a fallacy in exploring dilatation of the aorta, 451
———, general symptoms of, 508
———, treatment of, 288
Anæmic murmurs (see *Inorganic Murmurs.*)
——— in pregnancy, 134
(See *Utero-placental Murmur.*)
——— pulsation of the abdominal aorta, 464
Anatomy of the heart, 1
Andral, M., on depositions in arteries, 228
———, on hypertrophy, 247
———, on ossification, 363
———, on transformations of tissues, 359
Aneurism, abdominal, case of, 462
———, fallacies of, 459
———, signs of, 457, 462
Aneurism, thoracic, anatomy of, false, 422
———, mixed, 425
———, true, 421
———, bursting into the right ventricle, 466
———, bursting into the pulmonary artery, 469

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* Mr. James Freeman, to whom I am indebted for the following excellent Index says, "I have adhered strictly to the alphabetical order in all the divisions and subdivisions. Everything in an index is second to convenience, and convenience of reference is, in my opinion, only attainable by an unvaried alphabetical arrangement.

Aneurism, thoracic, classification of, 418
(See also *Dilatation of the Aorta.*)
————, causes of, 424
————, causing continuous murmur, 84
————, contents of the tumour, 426
————, convalescence from, 480
————, diagnosis from valvular disease, 442, 452
————, fallacies of, 455
————, from hernia of the internal membrane, 425
————, how do they cause erosion? 432
————, influence of sex in, 428
————, pathological effects of, 428
————, places where they burst, 430
————, prognosis of, 480
————, pulsation of, 439, 447, 453
————, purring tremor of, 446, 454
————, signs of, general, 435
————, signs of, physical, 441
————, situated behind the heart, 447
————, size of the tumour, 426
————, sounds of, first, 442, 444, 454
————, sounds of, second, 443
————, spontaneous cure of, 473
————, synopsis of signs of, 453
————, treatment of, by Albertini and Valsalva, 474
————, treatment of, recommended by the author, 477
————, perseverance in treatment of, 480
Aneurism of the ventricles, anatomy of, 321
————, causes of, 325
————, influence of age in, 326
————, influence of sex in, 325
————, pathology of, 326
————, signs of, general, 327
————, signs of, physical, 329
Aneurism of the auricles, 331
Angina pectoris (see *Neuralgia*)
Antacids, use of, in valvular disease, 415
Antispasmodics, use of, in valvular disease, 413
Aorta, abdominal, anæmic pulsation of, 464
————, treatment of, 481
————, pulsation of, from enteritis, 465
Aorta, thoracic, aneurism of (see *Aneurism*)
————, course of, 3
————, disease of the coats of, 222
————, dilatation of (see *Dilatation*)
————, forms a fixed point for the fibres of the heart, 58
————, inflammation of (see *Arteritis*)
————, obliteration of, 474
————, redness of its internal membrane, 203
————, roughness of, produces murmur, 72, 384
————, roughness of, produces tremor, 125
————, transformations of the coats of, 222
Apex, where it beats, 3
——, in dilatation, 294
——, in hypertrophy, 276
——, how it strikes the inferior margin of the fifth rib, and causes tinnitus, 41
(See cases of *Jones*, *Carrington*, *A . . . n, Esq.*, *Dr. A...... r.*)
Apoplexy, from diseased cerebral arteries, 224, 259
———— dilatation, 307
———— hypertrophy, 255, 266
———— obstructed circulation, 260
———— ossification, 258
Arterial thrill (see *Purring Tremor*)
Arterial varix, producing murmur and thrill, 107
Arteritis, acute, anatomy of, 220
————, signs of, 220
——, chronic, anatomy of, 221
————, a cause of depositions, 226
————, signs and treatment of, 231
Artery, compression of, causes murmur, 81, 104
——, depositions in the coats of, 222
————, causes of, 226
——, inflammation of (see *Arteritis*)
——, redness of the internal membrane, 203

Artery, suppuration of, 225
———, ulceration of, 225
Ascites, case of, causing murmur, 392
Asperity of the aorta, 72, 384
——— of valves, 72
Ass, experiments on (see *Experiments*)
Asthma, causes of, 392
———, ultimate cause of, 394, 397
———, from disease of the heart, 396
———, fit of, described, 401
—————, premonitory signs of, 402
—————, pulse in, 403
—————, subsidence of, 404
—————, modified by sleep, 399
—————, varieties of, 400
—————, why worse at night, 402
Atrophy of the adipose tissue of the heart, 348
——— of the heart, 356
——— of the valves, 73, 77, 364
Attenuation, how it modifies the sounds, 313 (See *Dilatation.*)
Attrition sounds in pericarditis, 166
—————, diagnosis of, from valvular murmurs, 174
Auricles, aneurism of, 331
———, dilatation of, 297, 298
—————, general signs of, 309
—————, physical signs of, 315
——— form a fulcrum beneath the ventricles, 58
———, hypertrophy of, 241, 249
—————, general signs of, 270
—————, relative frequency of, 244, 251
———, never empty, 24
———, repose of, its duration, 60
———, sinuses of, always full, 58
———, situation of, 4, 58
———, systole of, described, 55
—————, attended with retraction, 23
—————, does it cause an impulse? 55
—————, inconsiderable, 18, 23
—————, increased by palpitation, 18, 55
—————, in small animals, 18
—————, objects of, 57
—————, precedes ventricular systole, 11, 15, 18, 22
—————, produces no sound, 24, 63

Back-stroke (see *Diastolic Impulse*).
Beat of the heart described, 55
Belladonna, applied to aneurism, 480
Belladonna, for angina pectoris, 501
Bellows-murmur of Laennec, 82
—————, Laennec's fallacies on, 382 (See *Murmur*).
Bloodletting, a cause of polypi, 532
————— in aneurism, 474, 477
————— dilatation, 319
————— hypertrophy, 282, 284 (See *case of Macearl*).
————— pericarditis, 184, 187, 190
————— rheumatism, 179
————— valvular disease, 405, 407
—————, reaction from, 476
Bones, erosion of, by aneurism, 432, 434
Brain, congestion of, in dilatation, 307
———, disease of arteries in, 224, 259
———, effects of hypertrophy on, 255, 266
———, effects of ossification on, 258
———, inflammation of, in pericarditis, 154, 162
Bronchitis, diagnosis of, from pericarditis, 163
———, murmur in, 392
———, purring tremor from, 437
—————, diagnosis of, 457
Bruit musculaire, probably only venous murmur, 118
—————, diagnosis from jugular murmur, 116
—————, how far it causes the first sound, 25, 45, 48, 117

Calcareous degeneration, 221, 223
—————, causing ulcer, 225
—————, of the muscular substance, 353
————— of the valves, 421
Cancer of the heart, 355
Carditis, combined with pericarditis, 159
———, partial, 197
———, general, 196
Carotid aneurism, diagnosis of, from aortic, 456
Cartilage, effect of aneurisms on, 433
Cases, 542
Cartilaginous deposition on valves, 241
————— in the muscular substance, 353
————— in the pericardium, 354
Chlorosis (see *Anæmia*), 507
Chordæ tendineæ, shortening of, 77, 364
Clavicles, dislocations of, by aneurisms, 434
Cliquetis (see *Tinnitus*)
Colchicum, use of, in rheumatism, 179
Colon, tumor in, a fallacy in aneurism, 460
Columnæ carneæ in dilatation, 294

Columnæ carneæ in hypertrophy, 239
Communication of the two sides of the heart, 484
Complexion in dilatation, 306
——— in hypertrophy, 267
——— in softening, 337
Compression produced by aneurism, 429
Concretions in endocarditis, 211
——— causing murmur, 73, 531
——— caused by digitalis, 478, 532
———, intestinal, a fallacy in aneurism, 460 (See *Polypus*).
Configuration of an orifice modifies murmur, 83
Continuous murmur, 84
(See *Venous Murmur*, *Utero-placental Murmur*, *Pericarditis*, *Murmur*).
Contraction, hypertrophy with (see *Hypertrophy*)
Convulsions of the heart, 495
Costal percussion, sound of (see *Tinnitus*)
Cough in aneurism, 435
——— dilatation, 306
——— hypertrophy, 265
——— valvular disease, 374
Counter-irritation in endocarditis, 219
——— pericarditis, 187, 189, 190
——— valvular disease, 412
Current of blood, its strength modifies murmurs, 83, 84, 89
Cyanosis (see *Foramen Ovale*), 488
——— from perforation, 197
Cysts in the heart, 355

Deformity of the chest or spine modifies resonance on percussion, 8
Depositions in arteries, 223
———, causes of, 226
———, a cause of apoplexy, 258
———, from over-distension, 229, 259
Depth or hollowness of murmurs, 86
Destruction of parts by aneurism, 429
Diaphragm, tumor in, a fallacy in aneurism, 460
Diaphoretics in valvular disease, 410
Diastole (see *Ventricles*)
Diastolic impulse in hypertrophy, 67, 271
Diet in aneurism, 479
——— dilatation, 317
——— endocarditis, 219
——— hypertrophy, 282, 285
——— pericarditis, acute, 186
———, chronic, 190
——— valvular disease, 405, 416
Digitalis, use of, causes polypi, 478, 532
———, in aneurism, 478
———, in dilatation, 318
Digitalis, use of, endocarditis, 219
——— hypertrophy, 288, 290
——— pericarditis, 188
——— in valvular disease, 414
Dilatation, anatomy of, 294
———, auricular, signs of, 309, 315
———, causes of, 250, 298, 317
———, complicated with aneurism, 435
———, complicated with valvular disease, 373, 375
———, distinction of, from distension, 296
———, extent of the sounds in, 312
———, formation of, 297
———, impulse in, 68, 310
———, partial (see *Aneurism of Ventricles*)
———, pathological effects of, 299
———, prognosis of, 316
———, progress and terminations of, 315
———, pulse in, 305
———, regurgitation and murmur from, 77, 308
——— of the right ventricle, signs of, 308
———, signs of, general, 305
———, signs of, physical, 309
———, sounds in, 68, 310
———, treatment of, 316
———, varieties of, 293
Dilatation of the aorta, (see also *Aneurism*,) 418
———, anatomy of, 419
———, formation of, 419
———, pathological effects of, 428
———, purring tremor from, 125, 446
———, seat of, 419
———, sounds in, 72, 445
———, signs of, 451
———, treatment of, 480
Dilatation of the pulmonary artery, 420
———, signs of, 452
Displacements of the heart, 536
Distension of arteries, a cause of depositions, 229
——— the heart, 297
———, distinguished from dilatation, 296
——— differs from fulness, 21, 57
Diuretics, use of, in dilatation, 319
——— hypertrophy, 287
——— pericarditis, 189
——— valvular disease, 408
Dogs, experiments on, illustrative of inorganic murmurs, 100

Dropsy in aneurism of the heart, 328
——— in dilatation, 300, 306
——————, treatment of, 319
——— in hypertrophy, 252, 254, 267
——————, treatment of, 287
——— in pericarditis, 189
——— in valvular disease, treatment of, 408, 411
Dulness on percussion, natural extent of, 7
——————, causes counteracting, 8
—————— in aneurism, thoracic, 440
—————— in aneurism, abdominal, 458, 461
—————— in dilatation, 7, 314
—————— in endocarditis, 215
—————— in hydropericardium, 7, 539
—————— in hypertrophy, 276
—————— in pericarditis, 161, 164
—————— in peripneumony, 163
—————— in pleurisy, 163
Dura mater, ossification of, 226
Dyspepsia producing palpitation, 505
——————, cases of, 514, 521
Dyspnœa in aneurism of the aorta, 435, 438
——— in aneurism of the heart, 528
——— in dilatation, 306
——— in endocarditis, 213, 215
——— in hypertrophy, 252, 264
——— in pericarditis, 156, 158
——— in valvular disease, 374

Effusion in pericarditis, 152
——————, absorption of, 181
——————, signs of, general, 158
——————, signs of, physical, 164
Elasticity of the heart, what, 1
——— of arteries, loss of, 222
Emetics, use of, in valvular disease, 410
Emphysema of the lungs modifies dulness, 8, 276, 314
(See case of *Keith*).
Endocarditis, anatomy of, 171, 201, 203
———, causes of, 217
———, causing transformations, 354
———, causing vegetations, 369
———, coagulation of blood from, 211 (See case of *Feun*).
———, complicated with pericarditis, 151, 171, 212
———, dulness in, 215
Endocarditis, duration of, 217
———, impulse in, 216
———, gangrene from, 210
———, lymph effused in, 208
———, pain in, 381
———, prognosis of, 218
———, progress of, 217
———, pulse in, 213, 214
———, redness of, 203, 208
———, signs of, general, 213
———, signs of, physical, 171, 215
———, sounds in, 216
———, summary of signs of, 217
———, terminations of, 218
———, treatment of, 219
———, ulceration from, 210
Enteric inflammation, a cause of ventral pulsation, 465
Excito-motory system, is it the agent of the heart's movements? 66
——————, the seat of asthma, 394
Expectorants in valvular disease, 412
Experiments on the rabbit and frog, 11
——— on the ass, first series, 14
——————, second series, 17
——————, repeated Aug. 10, 1831, 20
——————, performed Nov. 3, 1834, 30
——————, performed Feb. 1835, 32
—————————, originality of, 30
——————, performed Aug. 7, 1835, 35
——— on dogs, illustrative of inorganic murmurs, 100
Expiration, effect of, on the impulse and sounds, 5
——————, on jugular turgescence, 270
——————, on the venous murmur, 114
Extension (see *Muscular Extension*)
External applications to aneurisms, 480
Eye, state of, in hypertrophy, 261

Fæces, indurated, a fallacy in aneurism, 460
False aneurism of the aorta, 418, 422
Fat, excess of, in the heart, 347, 352
——————, cases of, 348, 350, 351
Fever, pulse in, 201, 335
——, redness of the endocardium in, 206
——, rheumatic, (see *Rheumatism*)
——, symptomatic, of endocarditis, 213
——————, of pericarditis, 155
Fibrous deposit in arteries, 223
———, in the heart, 353
Filing murmur of Laennec, what, 82
Fœtal heart, beat of, characters of, 129

Fœtal heart, beat of, exploration of, 127
————————, fallacies of, 123
————————, imitated by jerking tape, 27
————————, speed of, 129
————————, time of the occurrence of, 128
————————, value of, 130
————————, where most audible, 129

Foramen ovale, patescence of, 484
————————, cases of, 491, 492
————————, general signs of, 487
————————, physical signs of, 489
Friction, the cause of murmurs and tremor, 80, 98
Fulness of the heart, differs from distension, 21, 57

Gangrene of the arteries, 221
——— of the heart, 199
——— from endocarditis, 210
——— of the legs from ossification, 224
Gases, use of, in valvular disease, 413
Glands, pulsating, a fallacy in aneurism, 455
Greasy degeneration of the heart, 348

Hæmoptysis in hypertrophy, 262, 265
Heart, acts when removed from the body, 58
——, adipose degeneration of, 347
——, anatomy of, 1, 235
——, aneurism of, 320
——, asthma from disease of, 396
——, atrophy of, 356
——, diastole of, (see *Ventricles*)
——, dilatation of, 293
——, dimensions of, 235
——, displacements of, 536
——, distension of, 296
——, first principle of the motion of, 65
——, fœtal, 127
——, form of the cavities of, 296
——, gangrene of, 199
——, greasy degenerations of, 347
——, hypertrophy of, 233
——, induration of, 346
——, inflammatory affections of, 143
——, lining membrane of, 358
——, malformations of, 482
——, motions of, described, 55
————————, causes, mechanism, and objects of, 57
——, nervous affections of, 495
——, organic diseases of, 232
——, ossification of, 353
——, pathological phenomena of the action of, 67
Heart, perforation of, 197
——, repose of, (see *ventricles*)
——, rupture of, 198, 295
——, situation of, 2
————————, when enlarged, 4
————————, how altered by position, 5
——, softening of, 332
——, sounds of, (see *Sounds*)
——, systole of, (see *Ventricles*)
——, weight of, 235
Hepatic artery, aneurism of, 460
Hernia of the internal membrane of arteries, 425
Hum, venous (see *Venous Murmur*) 118
Hydræmia (see *Anæmia*) 123
Hydropericardium, dulness in, 7, 539
————————, a fallacy in aneurism, 455
————————, signs of, 538
————————, treatment of, 539
Hypertrophy, anatomy of, 237
————————, of the auricles 241,
————————, of the ventricles, 240
——————, bloodletting in 284
——————, causes of, 242, 245
——————, classification of, 234
——————, complications of, 279, 375, 435
——————, counteracted by valvular disease, 261
——————, diet in, 285
——————, dulness in, 276
——————, formation of, 242
——————, from adhesion of the pericardium, 192
——————, history of, 233
——————, impulse in, 67, 270
————————, with dilatation, 69
——————, inflammatory, 246
——————, order in which the clavicles are affected by, 248
——————, pathological effects of 252,
————————, with contraction, 255, 263
————————, on the brain, 255
————————, on the lungs, 262
——————, prognosis of, 281, 289
——————, progress of, 277
——————, pulse in, 265
——————, relative frequency of forms and seats of, 251
——————, signs of, general, 263, 277
————————————, of the auricles, 270
————————————, of the right ventricles, 268
————————, physical, 270
——————, sounds in, 67, 274
————————, causes of their modifications, 68
——————, terminations of, 277

Hypertrophy, treatment of, 281
——————————, success of, 289
——————, valves in, 241
——————, with contraction, does it exist ? 291

Ice, application of, to aneurisms, 480
Iliac arteries, ossification of, 224
Impulse, caused by the ventricular systole, 19
————, circumstances which diminish the, 69
————, coincides with the systole, 18
————, in carditis, 197
————, in dilatation, 68, 310
————, in endocarditis, 213, 214, 216
————, in hydropericardium, 539
————, in hypertrophy, 67, 270
————————————, causes which diminish the, 274
——————————, with dilatation, 69, 272
————, in induration, 346
————, in pericarditis, 165
————, in softening, 339
————, jogging, 194, 449
————, mechanism of, 58
————, modified by posture, 5
Iuduration, anatomy of, 346
————, impulse of, 346
————, in hypertrophy, 237
————, proof of inflammation, 196
————, treatment of, 346
—————— of valves, aortic, 365
————————————, mitral, 362
————————————, pulmonary, 366
————————————, tricuspid, 366
Inequality of the pulse, what, 377
Inflammation, cause of ventricular aneurism, 325
————————————, depositions in arteries, 226
————————————, hypertrophy, 246
————————————, polypi, 529
————————————, softening, 332
————————————, valvular disease, 373
————————————, vegetations, 38, 369
————————, does it contribute to the effects of aneurisms, 433
Inorganic murmurs, 95
————————————— accompany arterial diastole, 98
————————————— accompany ventricular systole, 98
————————————, case of, by M. Bouillaud, 104
————————————, caused by motion of the blood, 98
————————————, diagnosis from valvular murmur, 389
————————————— illustrated by experiments on dogs, 100
Inorganic murmurs in pregnancy, 131, 134
————————————, Laennec's views of, 95
————————————, physical causes of, 102
———————————— produced by anæmia 106, 509
—————————————— arterial varix, 107
—————————————— compression, 104, 391
—————————————— loss of blood, 106
—————————————— nervous excitement, 107
Inspiration, effect of, on the impulse and first sound, 5
——————————————, on jugular turgescence, 270
——————————————, on the venous murmur, 114
Intermission of the pulse, what, 377
Iron, use of, in anæmia, 510
————, in dilatation, 318
————, in softening, 341
Irregularity of the pulse, what, 377
Issues, use of, in valvular disease, 412 102
Jogging impulse, from adhesion of the pericardium, 194
——————————, from aneurism, 449
——————————, from displacement, 449
Jugular vein, pulsating turgescence of, 268
——————————, simple turgescence of, 309
——————————, varix of, a fallacy in aneurism, 456

Key, or pitch of valvular murmur, aortic, 85, 86
——————————————, mitral, 86
——————————————, pulmonic, 85
——————————————, tricuspid, 86
—————————— venous murmur, 110

Lead, acetate of, in aneurism, 478
Leeches in aneurism, 480
———— in pericarditis, 184, 187, 189, 190
Liver, congestion of, in dilatation, &c. 307
————, enlargement of, a fallacy in aneurism, 459
Lungs, murmur in aorta from disease of, 391
————, situation of, over the heart, 4
————, sounds heard through the, 34, 36, 39, 313
————, disease of, causing asthma, 394
————, engorgement of, in hypertrophy, 254
————, engorgement of, in dilatation, 306
——————————————, in valvular disease, 375
Lymph effused in endocarditis, 208

Lymph effused in endocarditis, organization of, 211
———————— in pericarditis, 146, 181
————————————, signs of, 166

Magnet, use of, in neuralgia, 501
Malaria, a cause of neuralgia, 500
Malformations of the heart, (see *Foramen ovale*,) 482
Measurements of the orifices, 295
Melanosis of the heart, 355
Membrane, lining, of the heart, 358
Mercury, use of, in endocarditis, 219
————————, in pericarditis, 186, 189
————————, in rheumatism, 179
Mesentery, tumor of, a fallacy in aneurism, 460
Metallic cliquetis of Laennec, cause of, 41
(See cases of *Jones*, *Carrington*, *A....n*, *Esq.*, *Dr. A......r.*)
Migration of rheumatism, 178
Mixed aneurism of the aorta, 418, 425
Mucous membranes, injection of, 307
Murmur, anomalous cases of, 391
———, continuous, 84, 167
——— from aneurism, abdominal, 458
————————————, thoracic, 444
——— attrition, 166
————————, diagnosis from valvular, 174
———— compression, 33, 34, 50, 104
———— concretions in the heart, 73, 531
———— endocarditis, 171
———— hypertrophy with dilatation, 69 94
———— ovarian tumor, 105
———— pericarditis, 166
———— polypi, 73, 531
———— roughness of the aorta, 72
———— regurgitations, 34, 37, 38, 50, 71, 73
——, inorganic, (see *Inorganic Murmurs*,) 95
——, musical, (see *Musical Murmur*,) 86
——, utero-placental, (see *Utero-placental Murmur*,) 131
——, venous, (see *Venous Murmur*) 109
——, valvular, (see *Valvular Murmur*,) 70
Muscular extension, described, 42
———————— sound of, differs from bruit musculaire, 43
————————, how far it contributes to the first sound, 42
Musical murmur, 86
———————— generally from regurgitation, 88
————————, what indicated by, 89
————————, in the veins, 118
(See cases of *Milton*, *V......*, *Esq.*. *James*, *Tindall*, *Jones*.)

Narcotics, use of, in neuralgia, 500
————————, in valvular disease, 514
Nervous murmur, (see *Inorganic Murmur*,) 107
——— palpitation (see *Palpitation*)
——— pulsation of the abdominal aorta, 464
Neuralgia of the heart, or angina pectoris, causes of, 497
———, diagnosis of, 500
———, nerve affected by, 499
———, prognosis of, 500
———, symptoms of, 496
———, treatment of, 500

Œsophagus, compression of, by aneurism, 438
Omentum, tumor of, a fallacy in aneurism, 460
Ophthalmic artery, how affected by hypertrophy, 261
Opium, use of, in neuralgia, 500
———————— in rheumatism, 179
———————— valvular disease, 514
Orifices, measurements of, 295
Ossification an attendant on age, 258, 363
—————, arterial, 223
————————, causes of, 226
—————, effects of, on the brain, 258
—————, is it natural to old age ? 363
—————, of the dura mater, 226
—————, of the substance of the heart, 353
————— of the ophthalmic artery, 261
————— of the pulmonary artery, 598
————— of the pleura, 226
————— of the valves, aortic, 365
———————————— mitral, 362
———————————— pulmonary, 367
———————————— tricuspid, 367
—————, generally on the left side, 359
————————————, why, 361
————————, causes of, 367
Ovarian tumor, causing aortic murmur, 105
————————————, murmur mistaken for uterine, 139

Pain in aneurism of the aorta, 438
———————— of the heart, 328
——— dilatation, 308, 498
——— excess of fat, 347
——— endocarditis, 213
——— hypertrophy, 268, 498
————————, how relieved, 284
——— pericarditis, 155, 160
——— valvular disease, 381
(See *Neuralgia*.)
Palpitation, auricular systole increased by, 55
———, causes of, 503
———, definition and description of, 504

Palpitation, impulse diminished by, 504
————, in aneurism of the heart, 328
———— dilatation, 305
———— endocarditis, 213, 214
———— hypertrophy, 263
———— pericarditis, 156
———— interval of repose diminished by, 61
———— nervous, 504
——————, anæmic, 507
——————, cases of, 512, 521
——————, dyspeptic, 505
—————— from stimulant diet, 510
——————, plethoric, 511
————, pain occasioned by, 381
———— produces dilatation, 297
————, sounds diminished by, 504
Pancreas, enlarged, a fallacy in aneurism, 459
Paralysis of the heart, 214, 495
Percussion of the heart, mode of, 5, 7
——————, theory of, 6
(See *dulness on percussion.*)
Perforation of the heart, 197
Pericarditis, adhesion from, object of, 148
——————————, process of, 149
————, amelioration, signs of, 163
————, anatomy of, 144
————, causes of, 176
————, convalescence in, 188
————, diagnosis of, 163
————, effusion of, 152
————, endocarditis coexistent with, 154, 172,
————, impulse of, 165
————, lymph effused in, 146
————, muscular substance affected in, 154
————, partial, 150
————, percussion in, 164
————, prognosis of, 182
————, progress of, 180
————, recurrence of, 188
————, redness of, 144
————, rheumatic, 177
————, signs of, 154
——————, general, 155
——————, diversities of, 157
——————, physical, 164
————, sounds of, 165
——————, from attrition, 166
——————, from endocarditis, 171
——————, diagnosis of the two classes, 174
————, terminations of, 181
————, transformations of false membrane, 151
————, treatment of, 184
————, white spots from, 151
Pericarditis, chronic, anatomy of, 153
——————, signs of general, 174
Pericarditis, chronic, signs of, physical, 176
——————, treatment of, 189
Pericardium, adhesion of, (see *Adhesion*)
————, bursting of aneurisms into, 430
————, inflammation of, (see *Pericarditis*)
————, situation of, 4
——————, when full of fluid, 5
————, transformations of, 354
————, tumor in, causing murmur, 225
————, white spots on, 151
Peripneumony, diagnosis of, from pericarditis, 163
Peritonitis, aortic pulsations in, 465
Phlebitis, 221
Plates, Figs. 2 to 22, inclusive, 622
——, description of, 613
Plessimeter, 6
Pleura, ossification of, 226
Pleurisy,
———, complicating pericarditis, 159
———, diagnosis of, from pericarditis 162
Pneumopericardium, 540
Poisons, paralysis of the heart by, 214, 495
Polypus, anatomy of, 525
——————, unorganized, 526
——————, slightly organized, 526
——————, more completely organized, 528
———, causes of, 528
——————, inflammation, 529
——————, retarded circulation, 528
———, formation of, 528
———, nature of, 524
———, signs of, general, 531
——————, physical, 531
———, solubility of, 535
———, treatment of, 532
Posture, effect of, on anæmic murmur, 459
—————— aneurismal murmur, 458
—————— the first sound, 5
—————— impulse, 5
—————— second sound, 3
———, in acute pericarditis, 156
——— chronic pericarditis, 175
——— asthmatic fit, 401
Pregnancy, auscultation of, 127
(See *Fœtal Heart, Uteroplacental Murmur.*)
Prominence of the præcordial region, 238, 276
Pulmonary apoplexy from dilatation, 306
—————— hypertrophy, 262

Pulmonary apoplexy, from mitral contraction, 249
——— softening, 344
Pulmonary artery, compression of by tumor, 225
———, course of, 2
———, dilatation of, 420
———, signs of, 452
——— forms a fixed point for the fibres of the heart, 58
———, inflammation of, (see *Arteritis.*)
———, ossification of, 76, 225
———, sound high up the, 64
Pulsating glands, a fallacy in aneurism, 455
Pulsation, abdominal, from inflammation, 465
———, anæmic or nervous abdominal, 464
———, aneurismal, abdominal, 457
———, thoracic, 439, 453
———, in dilatation of aorta, 451
———, in dilatation of pulmonary artery, 452
Pulse, in anæmia, 107
——— aneurism of the aorta, 436
——— dilatation, 305
——— endocarditis, 213, 214
——— fever, 201, 335
——— hypertrophy, 265
——— pericarditis, 156, 161, 165
——— softening, 338
——— valvular disease, 375
———, aortic contraction, 378
———, aortic regurgitation, 379, 384
———, mitral, 376
——— of the right side, 380
———, jerking, 165, 107, 379, 384
———, rapidity of, in rabbits, 61
——— should be felt when exploring valvular murmurs, 92
Pulses, table of, in diseases of the heart, 623
Puncturing for dropsy, 411
Purgatives, use of, in aneurism, 477
——— hypertrophy, 286
——— pericarditis, 185
——— rheumatism, 179
——— valvular disease, 409
———, influence of, on the dulness of abdominal aneurism, 477
Purring Tremor accompanies arterial diastole, 98
——— ventricular systole, 98
———, causes of, 98, 101, 125, 126
Purring Tremor, experiments on dogs, 100
——— in aneurism, 437, 446, 454
——— anæmia, 100, 106
——— arterial varix, 107
——— aortic dilatation, 451
——— compression, 104
——— endocarditis, 213
——— pericarditis, 165, 166, 174
——— pulmonary dilatation, 452
——— valvular disease, aortic, 385
———, mitral, 38, 125, 387
———, pulmonary, 386
———, tricuspid, 125
———, Laennec's views on, 97
———, synopsis of phenomena of, 124
———, venous, 605
(See case of *Pyke, Miss N . . . s.*)
Pus effused in arteritis, 223
——— pericarditis, 153
Putrefaction resembling gangrene, 199

Quiescence, necessity of, in aneurism, 479

Rasping murmur of Laennec, what, 82
Redness, in endocarditis, 201
——— inflammatory, 208
———, non-inflammatory, 203
——— pericarditis, 144
———, its value as a sign of inflammation, 206
Regurgitation, murmur from, 34, 37, 38, 50, 71
———, cause of, may be slight, 97
——— indicates organic disease, 89
———, from shortening of the chordæ tendineæ, 77, 364
———, from enlarged orifice, 74, 77, 97, 308
———, rules for exploring, 92
———, aortic, 73, 384
———, character of its murmur, 74
———, frequent occurrence of, 75
——— from aneurism, 74
——— from pericarditis, 165, 171
———, key of its murmur, 86
———, murmur of, where best heard, 74, 90
———, prolongation of its murmur, 74
———, pulse of, 379, 384, 466

Regurgitation, mitral, 76
————, key of, 86
————, pulse of, 376
————, recovery from, 308
————, where best heard, 91
———, pulmonary, 75
————, key of, 86
————, pulse of, 380
————, where best heard, 90
——, tricuspid, 79
———, key of, 86
———, pulse of, 380
———, where best heard, 92
Repose, interval of, 56
——, duration of, 20, 60
——, imperceptible during rapid action, 61
——, incompatible with distension, 60
——, object of, 60
——, omitted in Majendie's Theory, 61
Respiration, modifies impulse, 5
———— jugular turgescence, 270
———— sounds, 5
———— venous murmur, 114
—— suspension of, its effects, 23, 24, 70, 216, 396
Resonance on percussion, theory of, 6
Rheumatism, cause of endocarditis, 218
——, cause of pericarditis, 161, 177
——, preceding arteritis, 222
——, treatment of, 179
Rhythm of the heart, 20, 57
—— degrees of derangement of, 377
Ribs, erosion of, by aneurism, 434
——, relation of, to heart and vessels, 3
Roughness of murmurs, on what depending, 82
——, presumptions afforded by, 84, 89
Rowing, excessive, a cause of hypertrophy, 245
Rupture of the heart, 198, 322
———— valves and aorta, 199

Sawing murmur of Laennec, what, 82
Scale, Laennec's, of the extent of the sounds, 312
Serous infiltration in dilatation, 267
———— hypertrophy, 306
Setons, use of, in valvular disease, 412
Shock felt with the diastole, 37, 67, 271
———— systole, 33, 37, 43
Softening, anatomy of, 332, 336
——, cases of, 341
——, causes of, 332, 337
——, diagnosis of, 339
——, nature of, 332
——, proof of inflammation, 196
——, prognosis of, 340
——, pulse of, 339
Softening, red, 333
——, signs of, general, 338
————, physical, 339
——, supposed to be gangrene, 199
——, treatment of, 340
——, varieties of, 333
——, white, 336
——, yellow, 335
Sound, First, audible through lung, 34, 36, 39
——, auricular valves contribute to, 33, 38, 45
——, bruit musculaire contributes to, 45, 47, 63
——, causes of, 19, 25, 42, 50, 63
——, coincides with systole, 15, 17, 21
——, double, 41 602
——, muscular extension contributes to, 42, 63
——, not occasioned by impulse, 39
——, supposed to be imitated by contraction of the abdominal muscles, 48
——, where loudest, 27, 32, 37, 64
—, second, cause of, 19, 27, 33, 34, 37, 50, 63, 469
——, coincides with diastole, 17, 22, 24
——, imitated by jerking tape, 27
——, where loudest, 3, 34, 64, 275
Sounds, described, 9
——, diminished by compression, 450
——, do they differ on the two sides? 46, 311
——, duration of, 20
——, extent of, 311
—— heard at a distance from the patient, 354
—— heard through fluid, 39
—— heard through condensed lung, 313
—— in aneurism, 442
——— dilatation, 46, 63, 68, 310
——— endocarditis, 171, 216
——— excess of fat, 347
——— hydropericardium, 539
——— hypertrophy, 67, 274
——— hypertrophy with dilatation, 69, 275
——— pericarditis, 165
——— polypus, 531
——— softening, 339
——— valvular disease, 70, 382
Spasm of the heart, 495
Spots, opake, white, on the heart, 151
Stags, arterial ossification in, 229
Staybones, pernicious effect of, 246
Steatomatous disease of arteries, 221, 223
Sternum, relation of objects behind the, 3

Stethoscope, 93
Stomach, schirrus of, fallacy in aneurism, 459
Subclavian aneurism, diagnosis from aortic, 456
Syncope, 522
———, treatment of, 523
Systole, (see *Ventricles*)

Tape worms, mass of, fallacy in aneurism, 460
Tartar emetic, use of, in pericarditis, 186
Theories, erroneous or defective, on the sounds of the heart, 51
Thrill (see *Purring Tremor*)
Throbbing (see *Pulsation*)
Tinnitus, metallic, of Laennec, its cause, 41
(See case of *Jones*, *Carrington*, *A...n*, Esq., *Dr. A......r*)
Tonics, use of, in dilatation, 318
————— valvular disease, 416
Trachæa, compression of, by aneurism, 438
Transformations of Tissues, 230, 353, 359
—————, M. Andral on, 228
True aneurism of heart, 418, 421
(See *Aneurism of the Ventricles*)
Tubercles, case of, causing aortic murmur, 391
——————, referred to, 105
———— in the heart, 355
——————, case of, 608

Ulcers in the heart, 197
——————, signs of, 198
——— in arteritis, 225
———— endocarditis, 210
Utero-placental murmur, 131
————————, after fœtal expulsion, 140
————————, arterial whiff, 134
————————, cases of, 134
————————, characters of, 131
————————, continuous, 133, 135, 142
————————, is it sealed in uterine arteries? 141
————————, independent of pregnancy, 139, 142
————————, practical conclusions on, 142
————————, propositions respecting, 133
————————, simulated by tumors, 139
————————, situation of, 132
————————, time of occurrence of, 131

Valves, anatomy of, in health, 358
———, auricular, retropulsion of, contributes to the impulse, 59
———, situation of, auricular, 4
——————————, semilunar, 3
———, what included under the term, 45
Valvular disease, 358
————————— aggravated by hypertrophy and dilatation, 371
—————————, anatomy of, 358
——————————————, aortic, 365
——————————————, mitral, 362
——————————————, on the right side, 366
—————————, asthma from (see *Asthma*.)
—————————, causes of, 367
—————————, fallacies of, 391
—————————, influence of age on, 363
—————————, pain of, 381
—————————, pathological effects of, 371
—————————, prognosis of, 382, 406
—————————, progress of, 381
—————————, pulse of, 375
————————————, aortic contraction, 378
————————————, aortic regurgitation, 379
————————————, mitral, 376
————————————, of the right side, 380
————————, seat of, 359
————————, signs of, general, 374
————————————, physical, 382
————————————, aortic contraction, 383
————————————, aortic regurgitation, 384
————————————, mitral, 387
(See case of *Goff*)
————————————, pulmonary, 385
(See case of *Bowden*, *Rogers*)
————————————, tricuspid, 388
(See *Valvular Murmurs*)
————————, signs of arterial and auricular combined, 388
————————, terminations of, 382
————————, treatment of, 405
——————————————— antispasmodics, 413
——————————————— bloodletting, 407
——————————————— counter-irritation, 412
——————————————— diaphoretics, 410
——————————————— diet, 416
——————————————— diuretics, 408
——————————————— emetics, 410
——————————————— expectorants, 412
——————————————— gases, 413
——————————————— puncturing, 411
——————————————— purgatives, 409

Valvular disease, treatment of stomachics, 415
———— tonics, 416
————, why generally on the left side, 361
Valvular murmurs, 70
————, conclusions on, 89
————, configuration of orifice modifies, 83
————, depth or hollowness of, 86
————, diagnosis from attrition, 174
————, diagnosis from anæmia, 389
————, diagnosis from aneurism, 442
————, diastolic; aortic, 34, 73, 384
————; mitral, 78
(See case of *Goff*)
————; pulmonic, 37, 50, 76
————; tricuspid, 80
————, exploration of, 92
————, key of, 85, 89
————, mechanism of, 80
————, musical (see *Musical Murmur*)
————, pitch of, 85, 89
————, practical rules for exploring, 92
————, rough, on what depending, 82
————, presumptions afforded by, 84, 89
————, systolic; aortic, 33, 34, 50, 72
————; mitral, 50, 76
————; pulmonary, 33, 34, 50, 75
————; tricuspid, 50, 79
————, varieties of, 82
————, where best heard, semilunar, 90
————, auricular, 91
Varix, arterial, causing murmur and thrill, 107
——, jugular, a fallacy in aneurism, 456
Vegetations, anatomy of, 368
————, causes of, 369
————, globular, of Laennec, 527
Vegetations, nature of, 39, 210, 369
————, seat of, 368
(See *Valvular Disease.*)
Venous hum, 118
Venous murmur, 109
———— attends inorganic murmurs, 389
————, causes of, 122
————, described, 110
————, distinction from bruit musculaire, 116
————, existing in health, 124
———— during pregnancy, 133
————, modified by pressure, 111
———— respiration, 114
————, musical, 118
————, proved to be venous, 119
————, physical causes of, 121
————, obstructions to, 113
————, produced by tension, 115
————, situations of, 113
Venous thrill, 605
(See case of *Pyke, Miss N . . . e.*)
Ventricles, diastole of, 19, 56
————, causes of, 59
————, coincides with second sound, 22, 56
————, duration of, 20
————, never empty, 19, 21
————, repose of, 16, 19, 21, 56
————, duration of, 20, 60
————, object of, 60
————, situation of, 3, 4
————, systole of, 11, 15, 19, 56
————, causes which increase, 59
————, coincides with pulse, impulse, and first sound, 21, 56
————, duration of, 20
————, mechanism of, 58
Vertebræ, effects of aneurisms on, 433, 438

Warts (see *Vegetations*)
Weight of the heart, 237
Worms in the heart, 355

THE END.

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